AURICULAR FLUTTER

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PREFACE

Modern methods of clinical investigation have afforded much information regarding the nature and the causes of heart failure. This grave, and often fatal, event is now recognised as being not infrequently a result of disease of the auricles.

In order to understand the nature and significance of the disorders of the auricles, a preliminary account is given of certain facts concerning the anatomy and physiology of the heart, the pulsations of the jugular veins and the electrocardiogram.

The disturbances of the cardiac action that are due to extrasystoles and auricular fibrillation, and their relation to allied disorders, are discussed in the first chapter. The subsequent chapters deal with that condition of extreme acceleration of the auricles which is known as auricular flutter. The physiological, pathological, and clinical facts regarding this disorder, and the features which distinguish it from other disturbances of the heart's action, are described.

All the illustrations, with two exceptions, have been obtained from cases that have come under my own observation. To the *Edinburgh Medical Journal*, to the Clarendon Press, and to the publishers of *Heart*, I am indebted for permission to reproduce illustrations.

My sincere acknowledgments are rendered to my colleagues in the Royal Infirmary, and especially to Professor William Russell and Dr. Lovell Gulland, for their generous permission to examine and record the cases under their charge. To John Cowan, George D. Mathewson, and Theodore Shennan I am indebted for much kind assistance in the preparation of this work. I have to thank W. E. Hume and George D. Mathewson for allowing me to reproduce the records in Figs. 92 and 80, and W. Stevens for his skilled assistance in obtaining most of the electrocardiograms.

For the facilities afforded me for the investigation of cases I cordially express my indebtedness to the Trustees of the Clinical Medicine Research Laboratory of the Royal Infirmary and to the Laboratory Committee of the Royal College of Physicians of Edinburgh.

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May 1914.

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AURICULAR FLUTTER

CHAPTER I

INTRODUCTION

Nearly all our knowledge of the action of the auricles in disease has been acquired within recent years. It is true that the pulsations of the veins in the neck had been described by Galen [1] and by Morgagni [2]; nevertheless, in the middle of the last century, Stokes [3] admitted that "although in most cases of dilatation of the heart we find the auricles, as well as the ventricles, engaged, yet our knowledge of the disease as affecting the former cavities is very limited." He added that "fortunately this is not of much consequence to practical medicine." Graphic records of the venous pulsations in the neck were obtained by Bamberger,[4] Geigel,[5] and Marey [6] in 1863, and by Friedreich [7] in 1866. Riegel [8] in 1881 described certain features which were considered to distinguish the normal from the pathological venous pulse; and in Gibson's [9] paper in 1882 all the important facts that were then known regarding the action of the auricles in health and disease were discussed.

The publication of Mackenzie's [10] monograph in 1902 inaugurated a

[1] Galen, quoted by Morgagni.

[2] Morgagni, J. B., *De sedibus et causis morborum*, Venetiis, 1762, lib. i., Epist. anat. med., xviii., art. 9, 10, 11.

[3] Stokes, W., *The Diseases of the Heart and the Aorta*, Dublin, 1854, 273.

[4] Bamberger, H., "Beobachtungen über den Venenpuls," *Würzburger med. Zeitschr.*, Würzburg, 1863, iv. 232.

[5] Geigel, A., "Ueber den Venenpuls," *ibid.*, 1863, iv., 332.

[6] Marey, E. J., *Physiologie médicale de la circulation du sang*, Paris, 1863, 530-532.

[7] Friedreich, N., "Ueber den Venenpuls," *Deutsch. Arch. f. klin. Med.*, Leipz., 1866, i., 241.

[8] Riegel, F., "Zur Kenntniss von dem Verhalten des Venensystems unter normalen und pathologischen Verhältnissen," *Berl. klin. Wochenschr.*, 1881, xviii., 249.

[9] Gibson, G. A., "The Action of the Auricles in Health and Disease," *Edin. Med. Journ.*, 1882, xxviii., 118.

[10] Mackenzie, J., *The Study of the Pulse*, Edin. and Lond., 1902.

new era in our knowledge of the diseases of the heart. He showed how the jugular pulsations could be analysed with precision and accuracy, and moreover he proved that a careful analysis of tracings from these veins was of great clinical importance in determining the mechanism of the heart's action in health and disease. By means of the methods which Mackenzie introduced, an accurate clinical study of the auricles was, for the first time, rendered possible.

The next striking advance in our knowledge of the disorders of the heart's action was made by Wenckebach[1] in 1903. By a critical analysis of sphymographic tracings he demonstrated that each form of cardiac irregularity represented a disorder of one or other of the functional activities of the heart, namely, of stimulus production, excitability, contractility, and conductivity. This interpretation of the cardiac arrhythmias was adopted and amplified by Mackenzie[2] in 1908, and is now generally accepted.

The deeper insight that was gained by Mackenzie's methods of studying the heart disclosed the fact that many cardiac problems required re-investigation from the standpoint of anatomy, physiology, pathology, and therapeutics. Some of these problems have apparently been solved, while others still await elucidation. Within the last decade the most notable advance in the study of the heart has been effected by the introduction of Einthoven's[3] string galvanometer. By means of this instrument, as well as by the methods introduced by Mackenzie, it is now possible to study the action of the heart with a precision that was formerly unattainable, and the prognosis and treatment of cases of heart disease have consequently been placed on a more rational and scientific basis.

The Sinus Node

The "primitive tissue" in the auricular walls of the mammalian heart represents, according to Keith and Flack,[4] the remains of the

[1] Wenckebach, K. F., *Die Arhythmie als Ausdruck bestimmter Funktionsstörungen des Herzens*, Leipz., 1903; and English translation by Snowball, T., Edin., 1904.

[2] Mackenzie, J., *Diseases of the Heart*, Lond., 1908.

[3] Einthoven, W., "Ein neues Galvanometer," *Annalen d. Physik*, Leipz., 1903, 4 Folge, xii., 1059. See also papers in *Onderzoekingen gedaan in het physiologisch Laboratorium der Universiteit te Leiden*, Tweede Reeks, v., vi.

[4] Keith, A., and Flack, M., "The Form and Nature of the Muscular Connections between the Primary Divisions of the Vertebrate Heart," *Journ. of Anat. and Physiol.*, Lond., 1907, xli., 172.

FIG. 1.—Horizontal section (× 2 diameters) through the right auricle near its junction with the superior vena cava. *A*, the inter-auricular septum; *B*, right auricular appendix filled with thrombi; *C*, connective tissue in which there are many large nerves. *D*, the sinus node, in the right posterior aspect of the auricular wall, is demarcated by the lines drawn around it.

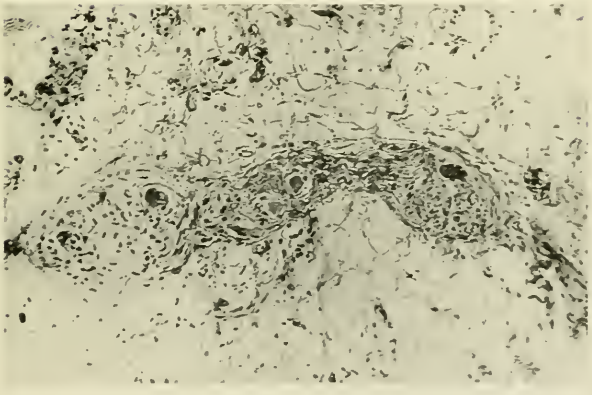


FIG. 2.—Nerve with ganglion cells near the sinus node. Lymphocytic infiltration of the connective tissue. Auricular flutter. Case III. (× 100 diameters.)

sinus venosus. This tissue may be found close to the mouths of the pulmonary veins and coronary sinus, and at one area where it is especially abundant it constitutes the sinus, or sino-auricular, node. This has the form of an elongated spindle, about 2–2·5 cm. in length and 2 mm. in breadth, which lies in the sub-epicardial tissue at the junction of the superior vena cava with the right auricular appendix (Fig. 1). The long axis of the node is parallel with the sulcus terminalis. If a section, in the long axis of the superior vena cava, be made through the cavo-auricular junction, the small artery running in the long axis of the node is seen in oblique or transverse section.

The sinus node consists of an irregular meshwork of slender muscle fibres, with definite transverse striation, embedded in somewhat compact connective tissue. These muscle fibres are in direct continuity with the adjacent muscle fibres of the auricular wall. Within the node, and running in its long axis, is the small artery already mentioned. In the vicinity of the node and elsewhere at the cavo-auricular junction there are abundant nerves and ganglia (Fig. 2). Some of the former pass into the node, but Miss Meiklejohn[1] found no nerve endings in the sinus node of man comparable to those in the sinus node of the monkey. The sinus node is generally held to set the pace and rhythm of the normal heart, and it is believed to be mainly through this node that vagus and sympathetic influences reach the auricles.

The Atrio-ventricular Conducting System

The system whereby stimuli are conducted from the sinus node and the auricles to the ventricles consists of a node, and a bundle with two branches and their terminal ramifications.

The atrio-ventricular node,[2] measuring about 2·7 by 1·7 mm., lies in the right lateral aspect of the auricular septum, in front of the mouth of the coronary sinus and above the posterior end of the septal cusp of the tricuspid valve. This node, like the sinus node, consists of an irregular meshwork of slender striated muscle fibres, but the amount of intervening connective tissue is less abundant. At the posterior extremity of the node its slender muscle fibres gradually merge in the larger muscle fibres of the auricular septum. A small artery usually passes forwards into the node.

[1] Meiklejohn, J., "On the Innervation of the Nodal Tissue of the Mammalian Heart," *Journ. of Anat. and Physiol.*, Lond., 1914, xlviii., 1.

[2] Tawara, S., *Das Reizleitungssystem des Säugetierherzens*, Jena, 1908.

The atrio-ventricular bundle,[1] which is about 1·5 mm. in diameter, consists mainly of a somewhat regular network of slender striated muscle fibres. It arises from the anterior extremity of the node and passes downwards, forwards, and to the left, until it reaches the lowest part of the pars membranacea septi (Plates II. and III.), where it divides. The atrio-ventricular node and bundle of man differ from those of most other mammalia in possessing a very scanty nerve supply. Miss Meiklejohn[2] was able to demonstrate a few small nerve fibres, but no nerve plexus or nerve endings.

The left branch of the bundle remains on the left side of the ventricular septum, and as its fibres pass downwards they spread out fanwise in the sub-endocardial tissue. The right branch, which is usually of ovoid or cylindrical form on transverse section, passes downwards and to the right until it lies in the sub-endocardial tissue on the right side of the ventricular septum. The right branch ultimately passes on to the moderator band of the right ventricle.

The terminal ramifications of both branches—the Purkinje fibres—run in the sub-endocardial tissue, and eventually merge with the ventricular muscle fibres. The Purkinje fibres which have the shortest course are those passing to the papillary muscles of the two ventricles.

By means of this system, stimuli from the sinus or auricles are transmitted to both ventricles simultaneously, and as the papillary muscles are the first to receive each stimulus, they begin to contract before the main mass of ventricular muscle.

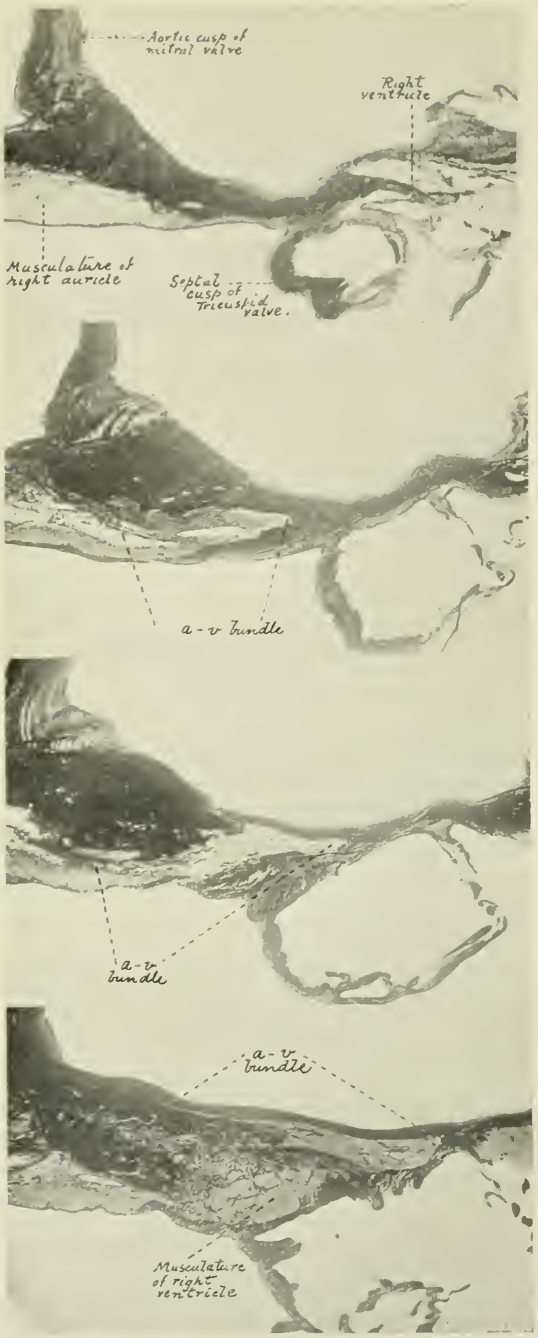
The Pulsations of the Jugular Veins

The rate and rhythm of the auricular contractions in health can usually be ascertained with considerable accuracy by careful inspection of the pulsations in the jugular veins. If the patient be in the recumbent posture, with his head low, gentle pulsation of the internal jugular vein is usually visible in the lower part of the neck, and is more distinct on the right than on the left side. In health, even although venous pulsation of considerable amplitude be seen, it cannot be felt when a finger is applied lightly over the vein, whereas firmer pressure

[1] Kent, A. F. S., "Researches on the Structure and Function of the Mammalian Heart," *Journ. of Physiol.*, Camb., 1893, xiv., 233. His, W., Jr., "Die Thätigkeit des embryonalen Herzens und deren Bedeutung für die Lehre von der Herzbewegung beim Erwachsenen," *Arbeiten a. d. med. Klinik*, Leipz., 1893, 14.

[2] Meiklejohn, J., *loc. cit.*

PLATE II.



Section No. 143.

No. 179.

No. 203.

No. 229.

FIG. 3.—Serial sections in the horizontal plane of the septum of a healthy human heart, illustrating the origin, position, and course of the atrio-ventricular bundle. The sections were treated with van Gieson's stain; the fibrous tissue consequently appears dark, the muscle tissue pale. (× 4.) (*Edin. Med. Journ.*, 1909, vol. ii.)

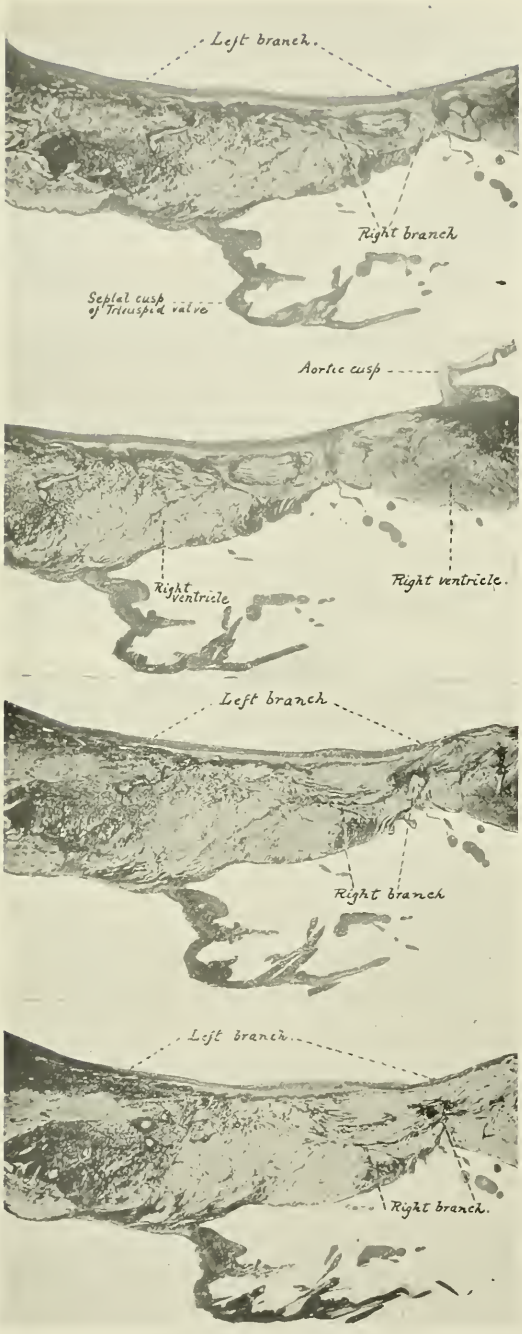


FIG. 4.—Further sections from the same heart as depicted in Plate II., to show the bifurcation of the atrio-ventricular bundle and its two main branches. (× 4.) (*Edin. Med. Journ.*, 1909, vol. ii.)

will enable the pulsation of the adjacent carotid, or subclavian, artery to be felt.

If the pulsations on the right side of the neck are watched, and if simultaneously their time relation to the apex-beat, the sounds of the heart or the carotid pulse on the left side, is noted, two or three impulses may be seen in the jugular vein for each beat of the ventricles. If only two impulses can be seen, the first is found to be ventriculo-systolic in time, for it appears to be synchronous with the apex-beat, the first sound of the heart or the carotid impulse, whereas the second impulse in the jugular vein occurs simultaneously with or immediately after the second sound, and is therefore ventriculo-diastolic in time. If a third impulse be visible in the veins, it is seen to occur immediately before the first sound of the heart is heard, and is therefore a pre-systolic impulse.

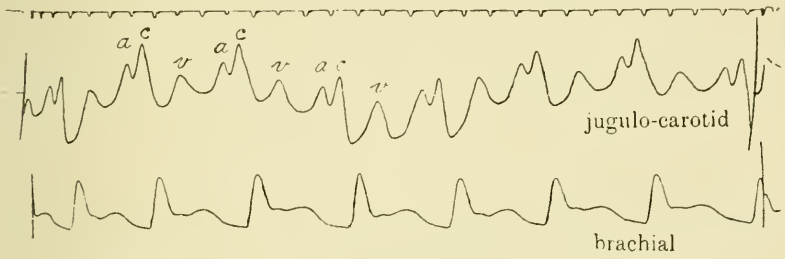


FIG. 5.—Normal jugulo-carotid pulsations. The time record is 0·2 second.

The pulsations in the vessels of the neck can be analysed with greater accuracy and precision if simultaneous tracings are taken from the jugular vein and from the apex-beat or the arterial pulse in the arm. The most serviceable instrument for this purpose is Mackenzie's[1] polygraph, which enables two tracings to be taken simultaneously, and also gives a time record in fifths of a second. Each rise of pressure in the auricles and in the jugular veins is represented by a rise on the jugulo-carotid tracing, whereas each fall of pressure within the auricles or great veins results in a depression of the tracing. Thus with each inspiration the intra-auricular pressure falls and the line of the tracing descends slowly; during each expiration the pressure in the auricles rises and the line of the tracing ascends gradually. In addition to these slow respiratory undulations, the tracing presents a series of quicker waves and depressions representing changes of pressure within the auricles during each cardiac cycle.

[1] Mackenzie, J., *Diseases of the Heart*, third edition, Lond., 1913, 105-108.

In analysing the jugular tracing, the time relation of its various waves and depressions to the events recorded in the arterial, or apical, tracing should be determined by means of careful measurement from the ordinates. It is then possible to ascertain the time in the cardiac cycle at which each wave or depression upon the jugular tracing occurs. For example, in the upper tracing of Fig. 6 the wave *c* begins 0·06 second before the commencement of the arterial pulse-wave in the lower tracing, and represents the carotid impulse transmitted to the jugular vein. Following the carotid wave there is a depression *x*. This is due partly to auricular relaxation, but also to the fact that during the early phase of ventricular systole the papillary muscles, pulling upon the auricles, enlarge these chambers and consequently lower the pressure within them, even although the auriculo-ventricular valves are closed. Towards the end of ventricular systole the intra-

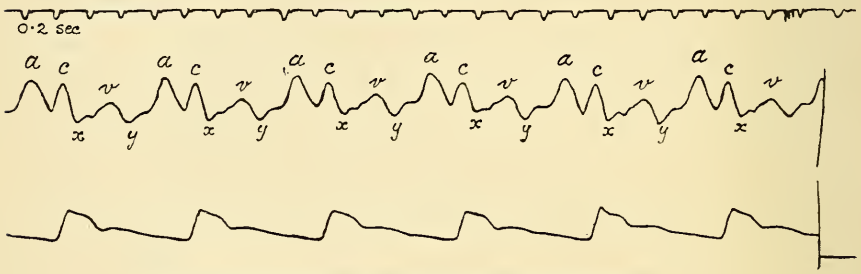


Fig. 6.—Normal rhythm. Jugulo-carotid and brachial tracings.

auricular pressure again rises, and the line of the jugular curve ascends gradually, so as to constitute the wave *v*. The pressure within the auricles and great veins continues to rise until the auriculo-ventricular valves open. This event is represented in the tracing by the summit of the wave *v*. The intra-auricular pressure then falls rapidly, as shown by the depression *y*, and thereafter the pressure rises slowly while both the auricles and the ventricles become filled with blood. About one-tenth of a second before ventricular systole, the auricles contract and the pressure within the auricles is notably increased. This is represented by the wave *a* in the jugular tracing.

The interval between the auricular and the carotid waves (the *a*-*c* interval) is appreciably longer than the auriculo-ventricular interval ($As - Vs = 0{\cdot}14$ second) because of the pre-sphygmic period and the time required for the transmission of the pulse-wave from the aortic orifice to the arteries of the neck. In health the *a*-*c* interval is about 0·17 second. Pathological prolongation of the *a*-*c* interval exceeding

0·2 second may be due to (1) delay in the transmission of the stimulus from auricle to ventricle; (2) lengthening of the pre-sphygmic period; (3) unduly slow transmission of the arterial pulse wave; or (4) any of these factors conjointly.

Although the normal jugulo-carotid tracing usually presents three waves *a*, *c*, and *v* in succession, certain other waves may be observed even when the heart is beating with a physiological rhythm.

(1) A small wave, *b*, may occasionally be seen interposed between the waves *a* and *c*. It probably represents an impulse transmitted to the blood within the auricle at the moment when the tricuspid valve closes early in systole.

(2) The wave *v* may be represented by two summits, v^1 and v^2. The notch or depression between them coincides approximately either

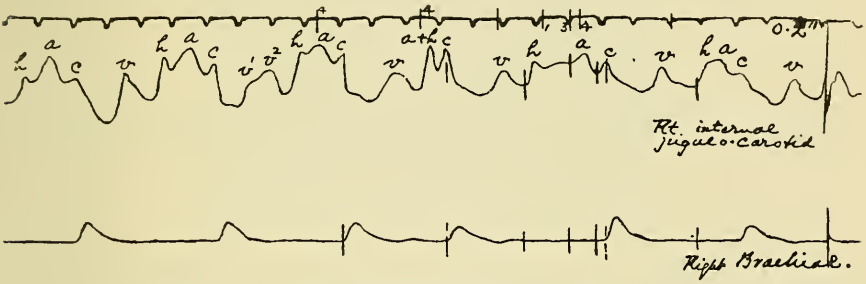


Fig. 7.—Sinus arrhythmia with *h* waves in the jugulo-carotid tracing. From a case of duodenal ulcer with healthy heart.

with the end of ventricular systole or with the closure of the semilunar valves.

(3) The wave *h* (Fig. 7), which was first described by Hirschfelder [1] and A. G. Gibson,[2] occurs in mid-diastole, and is ascribed to the apposition of the segments of the tricuspid valve when the ventricle becomes filled by the inrush of blood from the auricle. The interval between this wave and the antecedent carotid wave is constant for each individual patient. The *h* wave may be accompanied by an audible third heart sound.

The Electrocardiogram

Whenever a muscle or other excitable tissue is stimulated, changes in electric potential arise, and these can be recorded if a sufficiently

[1] Hirschfelder, A. D., "Some Variations in the Form of the Venous Pulse," *Bull. of the Johns Hopkins Hosp.*, Baltimore, 1907, xviii., 265.

[2] Gibson, A. G., "The Significance of a hitherto undescribed Wave in the Jugular Pulse," *Lancet*, Lond., 1907, ii., 1380.

sensitive galvanometer be used. The part of a muscle that is in contraction becomes electro-negative to the passive parts, and thus corresponds to the zinc plate of a galvanic cell, whereas the passive parts correspond to the copper plate. Waller,[1] in 1887, studied the action currents of the human heart by means of the capillary electrometer, and he showed that they could be led off from the skin surface; but it was not until 1903 that Einthoven[2] introduced the string galvanometer. It is this instrument, or one of its modifications, that is now used as the electrocardiograph in physiological and clinical laboratories.

The instrument consists essentially of an extremely fine fibre of

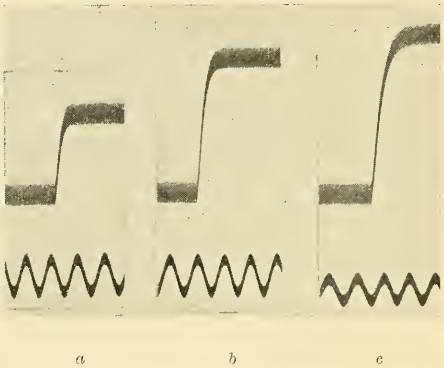


Fig. 8.—Control curves showing that the deflexion of the fibre becomes increased when the resistance is lowered, and that the deflexion time is increased when the tension of the fibre is diminished. In each record a difference of potential of 1 millivolt was introduced into the galvanometer circuit. In *a* and *c* the resistance was 1500 ohms greater than that in *b*. The deflexion time in *a* is 0·017 second; in *c* the tension of the fibre was relaxed, the deflexion is of greater amplitude, and the deflexion time is 0·046 second.

silvered glass or quartz, suspended between the poles of a powerful electromagnet and at right angles to the lines of force. Each end of this fibre can be connected to a non-polarisable electrode in which the patient immerses a hand or foot. The action currents of his heart, being thus led off from the skin, pass through the fibre which becomes deflected to one or other side according to the direction of the current passing through it. By means of an arc lamp, a substage condenser, a microscope, and a recording apparatus, the shadow of the moving fibre

[1] Waller, A. D., "A Demonstration on Man of Electromotive Changes accompanying the Heart's Beat," *Journ. of Physiol.*, Camb., 1887, viii., 229.

[2] Einthoven, W., "Ein neues Galvanometer," *Annalen d. Physik*, Leipz., 1903, 4 Folge, xii., 1059; "Die Konstruktion des Saitengalvanometers," *Arch. f. d. ges. Physiol.*, Bonn, 1909, cxxx., 287.

can be registered on a photographic plate or film. The record thus obtained is an electrocardiogram. The instrument is extremely sensitive, and if the fibre be properly adjusted it is absolutely aperiodic and has a deflexion time of about 0·02 second.

The amplitude of deflexion of the fibre is proportionate to the strength of current passing through it and to that of the magnetic field, and is inversely proportionate to the weight and tension of the fibre (Fig. 8). The strength of the magnetic field and the weight of the fibre are constant. The tension of the fibre can readily be standardised. For example, before each observation is made, the tension of the fibre is adjusted so that when the patient, or a resistance box substituted for him, is in the galvanometer circuit a difference of potential of 1 millivolt gives a deflexion of 1 cm. This standard, introduced by Einthoven, is generally adopted in electrocardiographic work.

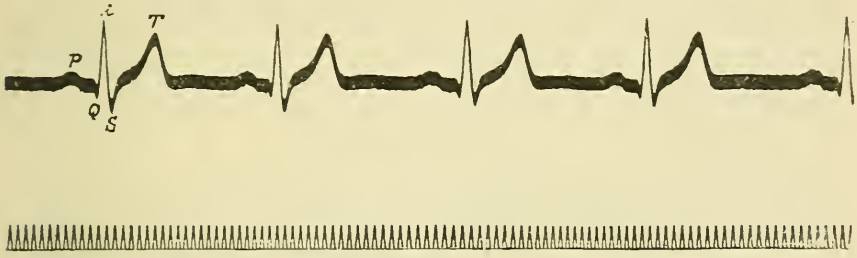


Fig. 9.—Normal electrocardiogram by derivation II. The time record is 28·57 per second.

The form of the electrocardiogram differs somewhat in any one individual according as the electrodes are applied to one or other part of the body surface. Following Einthoven,[1] we speak of derivation I. when the currents are led off from the right hand and left hand, and of derivations II. and III. when the right hand and left foot, and left hand and left foot, respectively are employed.

A normal electrocardiogram by derivation II. is shown in Fig. 9, while an electrocardiogram with a simultaneous record of the jugulo-carotid pulsations is shown in Fig. 10. The normal electrocardiographic deflexions are named *P*, *Q*, *R*, *S*, and *T*. Each upward deflexion signifies predominant negativity (activity) at the base or right side of the heart; each downward deflexion indicates predominant negativity (activity) at the apex or left side of the heart.

Auricular contraction is represented by the deflexion *P*, which, in

[1] Einthoven, W., "Weiteres über das Elektrokardiogramm," *Arch. f. d. ges. Physiol.*, Bonn, 1908, cxxii., 517.

health, is monophasic and directed upwards (Fig. 11, *a*). In some pathological instances the auricular deflexion, although monophasic, is directed downwards. Again, in some cases of auricular flutter and in some normal hearts under vagus inhibition the auricular deflexion becomes diphasic (Fig. 11, *b*). These abnormal auricular deflexions are

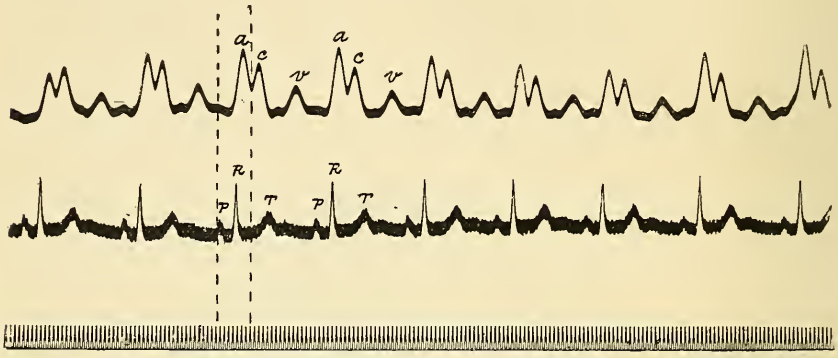


Fig. 10.—Jugulo-carotid pulsations and electrocardiogram by derivation II. The time record is 28·57 per second.

generally considered as evidence of the auricular contraction having started elsewhere than at the normal site (see also p. 93).

During the interval elapsing between the end of *P* and the commencement of the initial ventricular deflexion (*Q* or *R*), while the fibre is at rest, the stimulus is being transmitted along the atrio-ventricular

Fig. 11.—Diagrams of (*a*) monophasic, and (*b*) diphasic deflexions.

bundle to the ventricles. *Q*, *R*, *S*, and *T* are ventricular deflexions. Although their precise significance is still undetermined, we know that *R*, which is the most constant ventricular deflexion, precedes the apex-beat and the first sound by about 0·03 second (Kahn, Bull, Fahr, Lewis, Watson-Wemyss and Gunn[1]). The main mass of ventricular muscle

[1] Kahn, R. H., "Die Lage der Herztöne im Elektrokardiogramme," *Arch. f. d. ges. Physiol.*, Bonn, 1910, cxxxiii., 597; Bull, L., "On the Simultaneous Record of the Phono- and Electro-cardiogram," *Quart. Journ. of Exper. Physiol.*, Lond., 1911, iv., 289; Fahr, G., "On Simultaneous Records of the Heart Sounds and the

is in contraction during the interval *S-T*. The action currents probably compensate one another meanwhile, so that there is no predominant activity either at base or apex or in right or left ventricle. In the case of a healthy heart the terminal deflexion *T*, as recorded by derivation I., is directed upwards, and probably indicates predominant activity of the right ventricle over the left at the end of systole. In many cases of heart failure, *T*, by derivation I., is either of small amplitude or is directed downwards.

The particular value of an electrocardiogram is the information it yields regarding the sequence in which the various parts of the cardiac muscle pass into contraction. Contractions which originate at abnormal

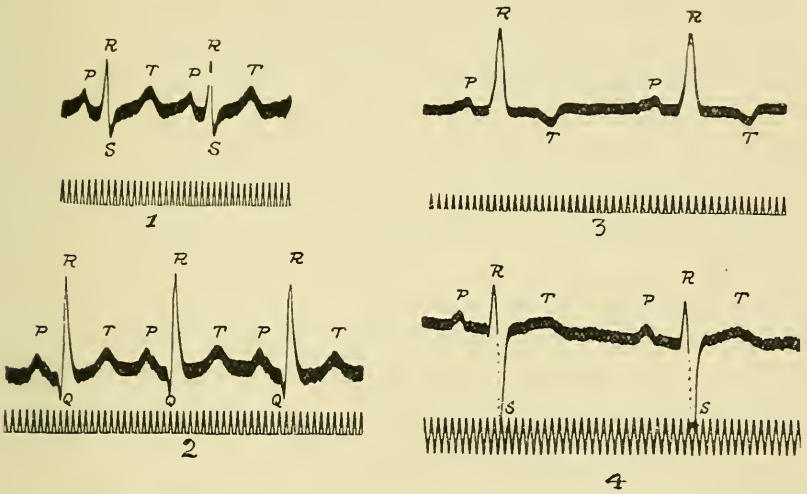


Fig. 12.—Electrocardiograms. I and 2 are by derivations I. and III. from a case of mitral stenosis; 3 and 4 are by derivations I. and III. from a case of aortic incompetence.

sites, or which spread through the heart in an abnormal manner, are represented by electrocardiographic deflexions of abnormal form. Moreover, the form of the ventricular deflexions may show departures from the normal according as the right or left ventricle is in predominant hypertrophy (Fig. 12), and also according as the vagus or the sympathetic fibres to the heart are stimulated. Not the least important advantage of the electrocardiograph over the polygraph is the ease wherewith the auricular contractions can be recorded and their nature,

Electrocardiogram," *Heart*, Lond., 1912-13, iv., 147; Lewis, T., "The Time Relations of Heart Sounds and Murmurs, with Special Reference to the Acoustic Signs in Mitral Stenosis," *Heart*, Lond., 1912-13, iv., 241; Watson-Wemyss, H. L., and Gunn, J. D., "Simultaneous Electro- and Phono-cardiograms," *Edin. Med. Journ.*, Edin., 1913 (N. S.), xi., 124.

whether normal or abnormal, can be demonstrated. Electrocardiograms are therefore of the utmost value in the recognition of all abnormal forms of auricular contraction.

Extrasystoles

It has long been known that if the heart be excised and placed under suitable conditions it may continue to beat rhythmically for hours. The site at which the stimuli for contraction are initiated is therefore within the heart itself. According to the myogenic theory, which is based in great measure on the epochal researches of Gaskell,[1] stimulus production and the conduction of stimuli from one part of the heart to another are inherent automatic functions of the heart muscle. According to the neurogenic theory, however, the stimuli for the heart's contraction are generated in, and conducted by, nerve elements. Whichever theory be correct, rhythmic stimuli are initiated in the healthy heart, the muscle fibres receive each stimulus, contract in response thereto, and maintain a certain degree of tone. The function of stimulus production is most highly developed in the sinus, whether it be the sinus venosus itself, or, as in the mammalian heart, the specialised tissue in the sinus node and in the ostial portions of the great veins. The sinus therefore sets the pace and rhythm of the whole heart. In health, the other chambers, being endowed with a less frequent capacity to initiate contractions, beat only when a stimulus from the sinus is transmitted to each of them in sequence.

Heart muscle differs from skeletal muscle in two fundamental respects. If a stimulus applied to the heart be strong enough to evoke a contraction, this is not proportionate to the strength of the stimulus but is always maximal. This phenomenon constitutes the "all or none" law of Bowditch.[2] Again, while the heart muscle is in contraction it is incapable of excitation—the heart is in the refractory phase. During diastole, excitability is restored gradually.

A response, in the form of a premature beat or extrasystole, is evoked if a stimulus of effective strength is applied to the heart muscle after its refractory phase has ended, and yet before the succeeding physiological stimulus is due. An extrasystole may therefore be defined

[1] Gaskell, W. H., "The Contraction of Cardiac Muscle," *Schäfer's Text-Book of Physiology*, Edin. and Lond., 1900, ii., 169-227.

[2] Bowditch, H. P., "Ueber die Eigenthümlichkeiten der Reizbarkeit, welche die Muskelfasern des Herzens zeigen," *Ber. über d. Verhandl. d. könig. sächs. Gesell. d. Wiss., Math.-phys. Classe*, Leipz., 1871, xxiii., 652.

as a premature beat of the auricles and ventricles, of auricles alone, or of ventricles alone, in response to a heterotopic stimulus, namely, to one that is initiated at some part of the heart other than the physiological pacemaker. If the excitability of some portion of the heart be unduly high, the application of a relatively weak stimulus may excite

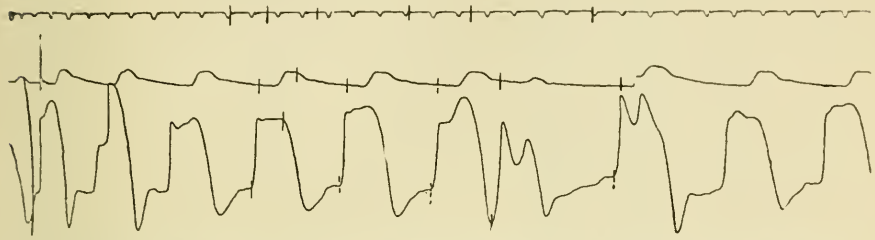


Fig. 13.—In the lower (apical) tracing the seventh beat is an extrasystole. In the upper (brachial) tracing the corresponding pulse-wave is small and delayed. From a man, aged 63, with aortic and mitral incompetence.

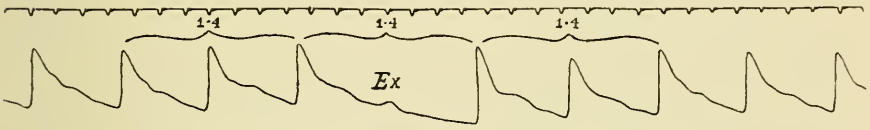


Fig. 14.—An extrasystole followed by a fully compensatory pause.

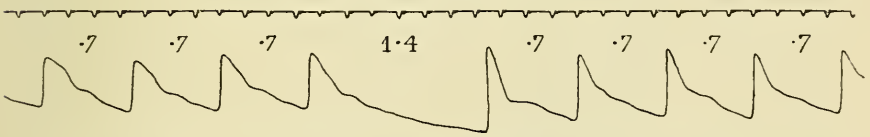


Fig. 15.—The extrasystole does not cause a pulse-wave, but the post-extrasystolic pause is fully compensatory.

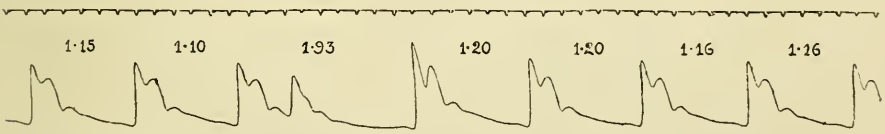


Fig. 16.—The extrasystole is followed by a pause that is not fully compensatory.

an extrasystole, whereas in the case of a healthy heart a stronger stimulus would be necessary.

The most obvious and constant character of an extrasystole is its premature incidence in the course of a series of rhythmic physiological beats. In some extrasystoles only the ventricles contract prematurely, the auricles meanwhile pursuing an unchanged rhythm, but in other instances both the auricles and ventricles contract prematurely. In either case the premature beat of the ventricles may open the semi-

lunar valves and thus transmit a pulse-wave into the arteries, or may fail to do so (Figs. 14, 15).

As was pointed out by Marey,[1] an extrasystole is usually followed by a diastolic pause—a post-extrasystolic pause—of longer duration than that after each physiological beat. In many instances the rhythm of the sinus is not disturbed by the extrasystole, and the post-extrasystolic pause is then exactly compensatory (Fig. 14). The subsequent ventricular beats therefore occur at precisely the same time as if there had been no extrasystole. In other instances, when the stimulus that excites the extrasystole travels back to the sinus and disturbs its rhythm, the post-extrasystolic pause is not fully compensatory (Fig. 16). In rare instances the pause after an extrasystole may even be shorter than that after each physiological beat, and if the post-extrasystolic pause is so short that the following beat is not retarded at all, the extrasystole is said to be interpolated (Plate V., Fig. 18).

Varieties of Extrasystoles.—Extrasystoles may start in any portion of the heart. Those that are held to originate in the remains of the sinus (sinus extrasystoles), in the auricles (auricular extrasystoles), and in the atrio-ventricular node and bundle (nodal extrasystoles) may be grouped together as supra-ventricular extrasystoles. Ventricular extrasystoles originate either in the ventricular muscle or in the branches of the atrio-ventricular bundle. The characters that differentiate supra-ventricular from ventricular extrasystoles are well defined and distinctive, but the differentiation of one variety of supra-ventricular extrasystole from another is more speculative.

A ventricular extrasystole can be recognised readily. The apical impulse is premature, and if the aortic valve be opened and a pulse-wave transmitted into the arteries, this pulse-wave is also premature (Fig. 13). The auricular rhythm is not disturbed (Fig. 17). The premature beat of the ventricles is usually coincident with a rhythmic auricular beat, and as the auriculo-ventricular valves are meanwhile closed and the auricles are therefore unable to drive onwards their contained blood, they propel it backwards into the great veins, and thus a large wave appears in the jugular veins. The post-extrasystolic pause is, as a rule, fully compensatory.

As contrasted with the electrocardiographic deflections *Q*, *R*, *S*, and *T* of a physiological beat, a ventricular extrasystole is represented by an "atypical deflexion" which is usually of diphasic form (Fig. 17).

[1] Marey, E. J., *Physiologie expérimentale, Travaux du Laboratoire de*, Paris, 1876, ii., 63-86.

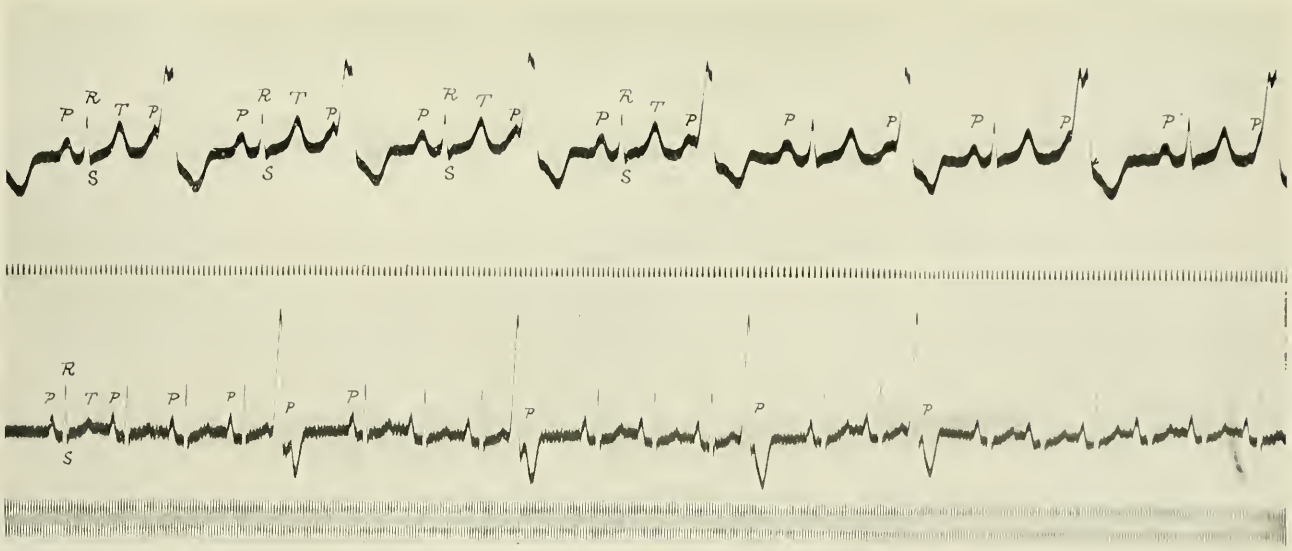


Fig. 17.—The upper record, from a woman aged 30 with mitral incompetence, shows a ventricular extrasystole after each normal beat. Derivation II. 1 cm. = 1 millivolt.
The lower record, from a woman aged 45 with carcinoma of the stomach, shows an occasional ventricular extrasystole. Derivation II. 1 cm. = 1 millivolt. In both, the time record is 28·57 per second.

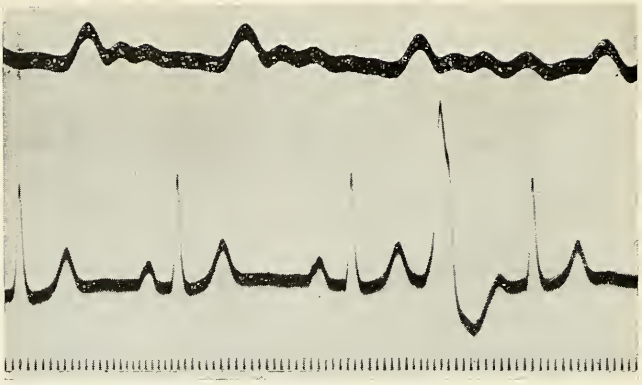
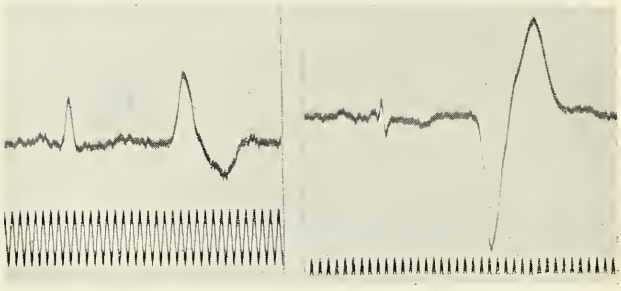
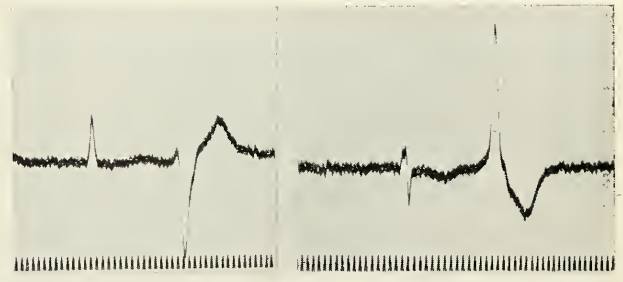


Fig. 18.—Sphygmogram and electrocardiogram by derivation II., showing an interpolated extrasystole. The inter-ventricular periods of the physiological beats are successively 0·80, 0·84, 0·84 second.



a. *b.*

Fig. 19.—Electrocardiograms; *a*, Derivation I.; *b*, Derivation III. Both show a normal beat and an extrasystole of type 1. From a man, aged 45, with aortic incompetence and heart failure, who died a fortnight later. 1 cm. =1 millivolt.



a. *b.*

Fig. 20.—Electrocardiograms; *a*, Derivation I.; *b*, Derivation III. In both records the second beat is an extrasystole of type 2. From a man, aged 69, with calcareous arteries, a systolic blood-pressure of 285 mm. Hg, dropsy, albuminuria, and auricular fibrillation. 1 cm. =1 millivolt.

This indicates that the various parts of the ventricular musculature are not passing into activity in normal sequence. The form of the deflexion varies according to the site of initial stimulation, whether on the right or left side, for example, and according to the derivation employed in recording the electrocardiogram. If the action currents are led off from the patient's left hand and left foot (derivation III.) the initial deflexion may be downwards and the terminal deflexion upwards (Fig. 19, *b*). In this case the extrasystole is held to arise in the apex or left ventricle, and possibly in the terminal fibres of the left branch of the atrio-ventricular bundle. If, with the same derivation (derivation III.), the initial deflexion is upwards and the terminal deflexion is downwards (Fig. 20, *b*), the extrasystole is regarded as arising in the base or right ventricle, and possibly in the terminal fibres of the right branch of the bundle. If, however, the records were taken by derivation I. (right hand and left hand), the form of the deflexions is usually the reverse of that by derivation III. Thus a left apical extrasystole by derivation I. yields a curve similar to that of a right basal extrasystole by derivation III. (Figs. 19, *a*, and 20, *b*). The former is the more common.

In a *supra-ventricular extrasystole* both auricles and ventricles contract prematurely, and as the rhythm of the sinus is disturbed the post-extrasystolic pause is not fully compensatory. If the auricles begin to contract before the ventricles (Fig. 21), the extrasystole probably originates either in the vicinity of the sinus node or in the auricular muscle. In the former case the auricular deflexion is of normal form. In many instances, however, the form of the auricular deflexion is abnormal, and it is often inverted, indicating that the auricular contraction began elsewhere than at the normal site. The ventricular beat usually yields a perfectly normal electrocardiogram, because the stimulus passes to the ventricles by means of the normal pathway, the atrio-ventricular conducting system.

In a *"nodal" extrasystole*[1] the auricles and ventricles contract prematurely and simultaneously. In some nodal extrasystoles the ventricles begin to contract before the auricles, but in others the auricles begin to contract about 0·06 second, or less, before the ventricles. But even in the latter instances, with an $As-Vs$ interval shorter than normal (0·14 second) the auricles are still in systole when the ventricles

[1] Mackenzie, J., "The Extra-systole: A Contribution to the Functional Pathology of the Primitive Cardiac Tissue," *Quart. Journ. of Med.*, Oxford, 1907-8, i., 131, 481.

begin to contract, because auricular systole lasts about 0·1 second. Whether the ventricles or the auricles begin to contract first, the fact of their being in contraction simultaneously suggests that the stimulus for their contraction is generated at a site more or less midway between them, and probably at the atrio-ventricular node or bundle (see also p. 115).

As a rule extrasystoles occur singly, but there may be multiple extrasystoles, one after another. Again, in any one patient all the extrasystoles are usually of one and the same variety, yield the same form of electrocardiogram, and presumably originate at the same site in the heart wall.

Although extrasystoles undoubtedly indicate an irritable focus in the heart, their clinical significance has been the subject of much controversy. Some authorities regard them as of little moment; others maintain that they indicate a definite, even though slight, impairment of the heart's functional efficiency. In my own experience, extrasystoles may be of purely nervous origin, but as a rule they have been associated with some insufficiency of the heart muscle. The defect may be slight and transient, as in the course of an acute infective disease. More often extrasystoles are either an early indication of arterio-sclerosis in an apparently healthy heart, or are associated with signs of more or less obvious heart failure, such as dyspnœa on exertion, precordial pain, and dropsy. In about 30 per cent. of my cases the larger arteries of the arm were notably thickened, and about the same percentage of cases presented evidence of mitral disease. Combined mitral and aortic lesions were found in 16 per cent.

From an analysis of 32 cases with supra-ventricular extrasystoles it appears that the prognosis in such cases is particularly unfavourable. Of these 32 cases, 7 subsequently developed persistent auricular fibrillation, and at least 10 of the 32 cases are known to have died within about two years after the extrasystoles were recorded. In some of these cases the fatal issue ensued in the course of a few weeks. For example, in a mason aged 49, a big, sturdily-built man, who had been subject to nocturnal attacks of precordial pain for two years, numerous auricular extrasystoles were recorded (Fig. 21). The patient was not dropsical, nor indeed was he so ill as to necessitate his being confined to bed. Two days after the last electrocardiogram, showing auricular extrasystoles, was recorded, he died suddenly. The post-mortem examination revealed chronic mediastino-pericarditis with chronic venous congestion of the liver, spleen and lungs, and in the lungs there was also much

PLATE VI.

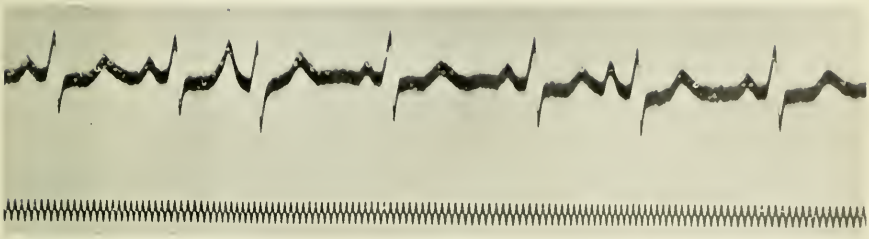


Fig. 21.—Electrocardiogram by derivation II. The third and sixth beats are auricular extrasystoles. In the former the auricular deflexion is merged with the terminal ventricular deflexion of the antecedent beat. From a case of syphilitic aortitis and myocarditis, three days before death.

Fig. 22.—Syphilitic aortitis.

fibrous induration as a result of silicosis. All the chambers of the heart were dilated, and all except the left ventricle were hypertrophied. The orifice of the left coronary artery was almost occluded by extensive syphilitic infiltration and scarring in the wall of the aorta (Fig. 22), and there were many fibrous patches in the wall of the left ventricle. The right auricle revealed only a slight degree of fibrosis without any leucocytic infiltration, while the atrio-ventricular node and bundle were healthy.

In this case the auricular extrasystoles were probably followed by auricular fibrillation, and the patient's sudden death may have been due to the ventricles subsequently passing into fibrillation in the manner suggested first by MacWilliam[1] and afterwards by Hering[2] and others. In this case, and in others where supra-ventricular extrasystoles were known to be the precursors of auricular fibrillation, with or without an intermediate stage of auricular flutter, the sequence of events suggests that the pathological changes in the walls of the auricles became progressively more intense and diffuse.

Auricular Fibrillation

When the co-ordinate contraction of the auricular musculature becomes replaced by inco-ordinate fibrillar contraction, the auricles dilate and are rendered functionally inactive in so far as they fail to expel their contained blood into the ventricles. Each ventricular contraction is still a co-ordinate one, but, as was shown experimentally by MacWilliam,[1] Philips,[3] and Fredericq,[4] the ventricular rhythm becomes wholly disorderly, and the rate of ventricular contraction is usually much accelerated. The suggestion made by Cushny and Edmunds[5] that complete irregularity of the ventricles in man might be due to auricular fibrillation was proved to be correct by Rothberger

[1] MacWilliam, J. A., "Fibrillar Contraction of the Heart," *Journ. of Physiol.*, Camb., 1887, viii., 296.

[2] Hering, H. E., "Ueber plötzlichen Tod durch Herzkammerflimmern," *Münch. med. Wochnschr.*, 1912, lix., 750, 818.

[3] Philips, F., "Les trémulations fibrillaires des oreillettes et des ventricules du cœur du chien," *Arch. internat. de physiol.*, 1904-5, ii., 271.

[4] Fredericq, L., "Rhythme affolé des ventricules dû à la fibrillation des oreillettes. Physiologie du faisceau auriculo-ventriculaire," *ibid.*, 1904-5, ii., 281.

[5] Cushny, A. R., and Edmunds, C. W., "Paroxysmal Irregularity of the Heart and Auricular Fibrillation," *Studies in Pathology*, edited by Bulloch, Aberdeen, 1906, 95; *Amer. Journ. Med. Sci.*, Philad., 1907, cxxxiii., 66.

and Winterberg[1] in 1909, and subsequently by the independent work of Lewis[2] and Jolly and myself.[3]

In the human heart auricular fibrillation is one of the most frequent forms of irregularity. It is decidedly more frequent in patients over forty years of age than in younger individuals. Males are twice as frequently affected as females, and in about one-half of the cases there is evidence of arterio-sclerosis. Fibrillation is especially common in cases of chronic interstitial myocarditis and in the terminal stages of mitral disease.

Auricular fibrillation may take the form of paroxysmal attacks

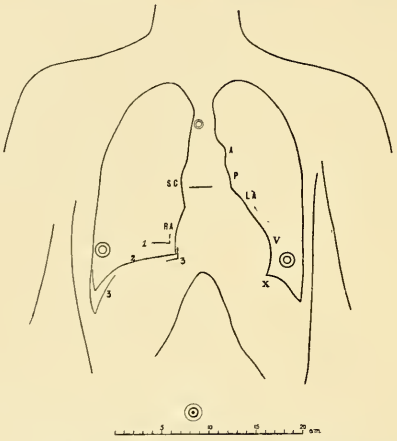


FIG. 23.—NORMAL HEART, FEMALE PATIENT, AGED 32.

1. In deep expiration. 2. In tranquil inspiration. 3. In deep inspiration.

A, Curve of aorta. *P*, Pulmonary curve. *LA*, Left auricular curve. *V*, Left ventricular curve. *SC*, Curve of superior vena cava. *RA*, Curve of right auricle. The position of the episternal notch, level of third chondro-sternal articulation, the nipples and umbilicus shown. *X* indicates the position of the apex-beat.

during which the ventricular rate may rise to 120-150, and in some cases even to 180-190 per minute. The initial attacks begin and end abruptly, but sooner or later fibrillation becomes persistent. The paroxysmal form was observed in a young woman aged 22, who was affected with mitral stenosis. When she was admitted to hospital, her pulse had a rate of 160-162 per minute. On the following morning

[1] Rothberger, J., and Winterberg, H., "Vorhofflimmern und Arhythmia perpetua," *Wien. klin. Wochenschr.*, 1909, xxii., 839; "Ueber das Elektrokardiogramm bei Flimmern der Vorhöfe," *Arch. f. d. ges. Physiol.*, Bonn, 1910, cxxxi., 387.

[2] Lewis, T., "Auricular Fibrillation and its Relationship to Clinical Irregularity of the Heart," *Heart*, Lond., 1909-10, i., 306.

[3] Jolly, W. A., and Ritchie, W. T., "Auricular Flutter and Fibrillation," *Heart*, Lond., 1910-11, ii., 177.

the normal rhythm was restored, and the rate was then 77 per minute. Fifteen months later this rhythm was still maintained.

A second example of paroxysmal fibrillation was that of an old gentleman who had enjoyed excellent health ever since childhood. At the age of 73, however, he had thickened arteries and an aortic systolic murmur. As a rule the rate of his pulse was about 78 per minute and its rhythm was perfectly regular except for an occasional extrasystole. His paroxysmal attacks of auricular fibrillation came on by day or by night, and usually lasted for several hours. Sometimes after having retired to bed feeling perfectly well, and having slept soundly for several hours, he would awake feeling weak and ill, and

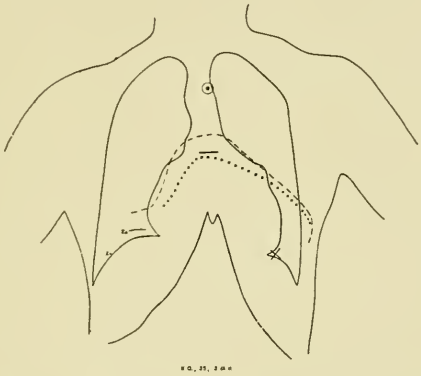


FIG. 24.—MITRAL STENOSIS, WITH AURICULAR FIBRILLATION, FEMALE, AGED 25.

A large globular heart.
- - - - - - The deep percussion dulness of the heart.
× × × × × × The superficial percussion dulness..

his pulse would then be extremely irregular and its rate about 160-170 per minute.

A third, and still more striking, case of paroxysmal fibrillation was that of a man aged 46, who had been ailing for a year and who, six months before I saw him, had been seized suddenly with what he called "a fluttering at the heart," accompanied by a violent paroxysm of coughing. He was an emaciated, anxious-looking man, whose clothes hung loosely on him, for he had lost 5 stones in weight. His heart was not enlarged and both sounds were pure, but as ascertained by means of the screen the aortic arch was dilated. When the cardiac rhythm was normal at the rate of about 100 per minute, he was liable to severe attacks of pain in the precordial region and left arm. Morphia alone afforded him relief. From time to time he suffered from sudden attacks

of auricular fibrillation, with a ventricular venous pulse and complete arrhythmia of the ventricles at a rate of about 120 per minute. During these attacks he felt the "fluttering," but never any pain. The attacks used to pass off spontaneously and suddenly, and they sometimes ceased while he was asleep.

He had been taking strophanthus for several months without obtaining real benefit. But within a fortnight of his commencing to take potassium iodide he was free of both the pain and the paroxysmal attacks of fibrillation, and was able to eat heartily and sleep soundly. His health was soon so far restored that he was able to travel to the

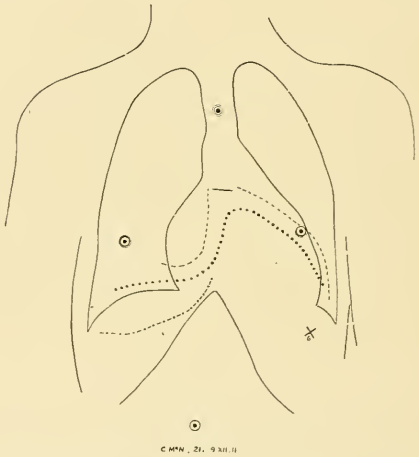


FIG. 25.—AURICULAR FIBRILLATION, MALE, AGED 21.

- - - - - - The deep percussion dulness.
× × × × × × The superficial percussion dulness in full expiration.
— · — · — · — The superficial percussion dulness in full inspiration.

south of England in search of sunshine and warmth. Two months later he died suddenly.

In a large number of cases, auricular fibrillation passes from the paroxysmal into the persistent form. The heart often becomes dilated and its outline more or less globular (Figs. 24 and 25). The transverse diameter of the heart becomes notably increased. In ten consecutive cases of fibrillation studied by means of orthoradiography, Hope Fowler [1] and the writer found the average maximum transverse diameter to be 15·4 cm., while the average long diameter, from the cavo-auricular junction to the apex, was 16·4 cm. In healthy adult males the average

[1] Fowler, W. Hope, and Ritchie, W. T., "Orthoradiography of the Heart and Aorta," *Edin. Med. Journ.*, Edin., 1912 (N. S.), ix., 197.

transverse and long diameters of the heart are about 12·1-13 cm., and 13-14 cm. respectively.

When the auricles are in fibrillation the efficiency of the heart as a pump becomes impaired and many of the classic symptoms and signs of heart failure usually supervene. As a rule, the intensity of the symptoms is proportionate to the rapidity of the ventricular rate. When this becomes retarded, as by means of digitalis, the urgent symptoms often vanish.

The jugular pulse in auricular fibrillation acquires the ventricular form. This is characterised by the absence of the auricular wave

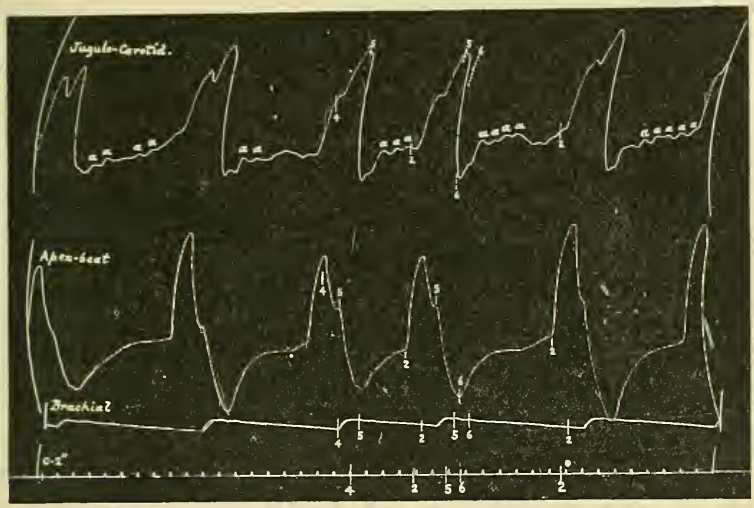


Fig. 26.—Auricular fibrillation. The jugular pulse is of the ventricular form, and during diastole small irregular wavelets are superposed on the curve. (*Heart*, vol. ii.)

immediately before each ventricular systole commences. The tracing rises abruptly with the onset of ventricular systole, and the systolic plateau, which may be interrupted by a depression due to the systolic pull of the papillary muscles, is maintained until the tricuspid valve opens. When this event occurs, and the blood pent up in the auricles rushes into the ventricles, the curve falls abruptly. Thereafter, it again ascends gradually while the pressure in the auricles and ventricles is rising during ventricular diastole. During this latter phase, small irregular wavelets of auricular origin may be superposed on the line of ascent (see Fig. 26).

Electrocardiograms do not present a normal auricular deflexion, *P*; but a series of small irregular deflexions at a rate of about 380 to 520 per minute is observed during ventricular diastole when the fibre

should be at rest (Fig. 27). These deflexions are most evident when the electrocardiogram is recorded by derivation III. (left hand and left foot). Although the ventricular rhythm is wholly disorderly, the ventricular deflexions are essentially of normal form, which indicates that these chambers are beating in response to stimuli of supra-ventricular origin.

Auricular fibrillation may be regarded as the combined result of two functional changes in the auricular muscle, namely, increased excitability and defective conductivity. It is not always possible to correlate these changes with structural alterations in the auricular muscle, yet inflammatory changes are usually found. Radasewsky[1] pointed out that persistent arrhythmia of the ventricles depends mainly upon diffuse interstitial myocarditis, affecting especially the right auricle. Sub-acute and chronic inflammation of the structures at the superior cavo-auricular junction has been described by Schönberg[2] and

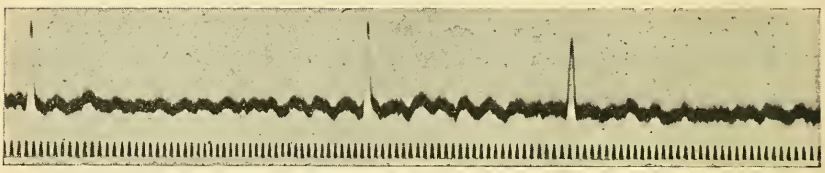


Fig. 27.—Auricular fibrillation with an infrequent ventricular rate. Derivation III. 1·5 cm. = 1 millivolt.

Hedinger,[3] while Koch,[4] Draper,[5] and others have recorded fibrosis of the sinus node. Six of my own cases presented diffuse fibrosis and extensive lymphocytic infiltration throughout the auricular walls, with similar lesions in the sinus node and in the atrio-ventricular conducting system. These structural changes, however, are not uniformly well marked.

Experimental observations indicate that extrasystoles, flutter, or

[1] Radasewsky, M., "Ueber die Muskelerkrankungen der Vorhöfe des Herzens," *Zeitschr. f. klin. Med.*, Berlin, 1895, xxvii., 381.

[2] Schönberg, S., "Ueber Veränderungen im Sinusgebiet des Herzens bei chronischer Arrhythmie," *Frankf. Zeitschr. f. Pathol.*, Wiesbaden, 1909, ii., 153; "Weitere Untersuchungen des Herzens bei chronischer Arrhythmie," *ibid.*, 1909, ii., 462.

[3] Hedinger, E., "Uber Herzbefunde bei Arrhythmia perpetua," *ibid.*, 1910, v., 296.

[4] Koch, W., "Zur pathologischen Anatomie der Rhythmusstörungen des Herzens," *Berl. klin. Wochenschr.*, 1910, xlvii., 1108.

[5] Draper, G., "Pulsus irregularis perpetuus with Fibrosis of the Sinus Node," *Heart*, Lond., 1911-12, iii., 13.

fibrillation can be induced according to the strength of stimulation applied to the auricular muscle; and clinical evidence demonstrates that auricular flutter may be preceded by supra-ventricular extrasystoles and followed by fibrillation. These three forms of abnormal auricular action may therefore be regarded as representing successive stages of abnormal stimulation of the auricles. When occasional supra-ventricular extrasystoles occur, it may be assumed that a single abnormal stimulus of effective strength is being generated, from time to time, at an irritable focus in the auricular walls. When multiple extrasystoles occur, the stimuli are probably being generated in rapid succession. When the auricles are in flutter, the abnormal stimulation of one focus in the auricular musculature is probably still more frequent and intense, leading to rhythmic co-ordinate contractions of the auricles at a rate that is greatly accelerated. Lastly, auricular fibrillation ensues when there is abnormal stimulation of great frequency and intensity at many foci, and when there is defective conduction of stimuli through the auricular walls.

CHAPTER II

ETIOLOGY

Auricular flutter is a pathological action of the auricles characterised by rhythmic co-ordinate contractions of their musculature at a rate that is greatly accelerated, and is usually between 250 and 300 per minute. The term "auricular flutter," as applied to the human auricles, was originally employed by Jolly and myself[1] in 1911. The condition has also been described under the designations "jugular embryocardia" (Morison),[2] "auricular tachysystole" (Rihl),[3] "auricular tachycardia" (Robinson),[4] and "auricular tachyrhythmia" (Hoffmann).[5]

In the preceding chapter it has been mentioned that an auricular extrasystole is induced when an effective stimulus is applied to the auricular muscle after its refractory phase has terminated, but before the next physiological beat is due. It has also been shown that when strong faradic stimulation is applied to the auricles, their co-ordinate contraction becomes replaced by fibrillation.

Auricular flutter, as a result of weak faradisation of the mammalian auricles, was first described by MacWilliam[6] in 1887. He stated that "the application of the current sets the auricles into a rapid flutter, the rapidity of which largely depends upon the excitability of the auricular tissue and the strength of current employed. The movements are regular; they seem to consist in a series of contractions originating in the stimulated area, and thence spreading over the rest of the tissue. The movement does not show any distinct sign of inco-

[1] Jolly, W. A., and Ritchie, W. T., "Auricular Flutter and Fibrillation," *Heart*, Lond., 1910-11, ii., 177.

[2] Morison, A., "Cardiac Motion as Revealed by the Vivisection of Disease," *Lancet*, Lond., 1909, i., 77 ; *ibid.*, 1909, i., 39.

[3] Rihl, J., "Hochgradige Vorhoftachysystolien mit Ueberleitungsstörungen und electiver Vaguswirkung," *Zeitschr. f. exper. Pathol. u. Therap.*, Berlin, 1911, ix., 277.

[4] Robinson, G. Canby, "The Influence of the Vagus Nerves on the Faradized Auricles in the Dog's Heart," *Journ. of Exper. Med.*, New York, 1913, xvii., 429.

[5] Hoffmann, A., *Die Elektrographie als Untersuchungsmethode des Herzens und ihre Ergebnisse*, Wiesbaden, 1914, 188-202.

[6] MacWilliam, J. A., "Fibrillar Contraction of the Heart," *Journ. of Physiol.*, Camb., 1887, viii., 296.

ordination; it looks like a rapid series of contraction waves passing over the auricular walls."

In a later paper (1888) MacWilliam[1] stated that when faradic stimulation of moderate strength is applied to the right or left auricular appendix "the auricles are thrown into a state of rapid fluttering action; and this movement continues during the passage of the exciting current and for a considerable time afterwards—the after-duration varying according to the excitability of the auricular tissue and the strength of the current employed. The individual contractions constituting the rapid fluttering auricular movement are weak and slight, as might be expected. Meanwhile the ventricles beat rapidly; much more rapidly than usual, though their action is not nearly so rapid as is that of the auricles. The ventricular acceleration appears to be due to the propagation of a more rapid series of beats from the auricles. All the auricular contractions indeed are not transmitted to the ventricles; often not more than one-half or even less are transmitted, but even such a proportion is sufficient to cause a very marked acceleration in the ventricular rate of beat. It is very possible that the weakness of the rapidly-recurring auricular contractions is an important factor in preventing a more complete propagation to the ventricles. The degree of acceleration that occurs varies considerably. In hearts beating at 140-180 per minute, stimulation of the auricular appendix often causes the auricular rate to rise as high as 500-600 per minute; meanwhile the ventricular rhythm may have a rapidity of from 200-300 per minute or even higher."

The clinical disorder that is designated auricular flutter is identical in all its essential features with the flutter induced experimentally by MacWilliam.

MacWilliam proceeded to describe the auricular flutter and ventricular responses upon stimulation of the "inhibitory area" of the mammalian heart. This area was described as a narrow, elongated area on the dorsal aspect of the auricles, overlying the lower part of the inter-auricular septum and its long axis running parallel with the plane of the latter. When this area of the auricular wall was stimulated, "the auricles are thrown into rapid fluttering movement much resembling that obtained by excitation of other parts of the auricular surface. At the same time the ventricles stand still in diastole and fill up with blood. This lasts for a considerable time while the rapid

[1] MacWillliam, J. A., "On the Phenomena of Inhibition in the Mammalian Heart," *Journ. of Physiol.*, Camb., 1888, ix., 345.

fluttering auricular action continues—stimulation of the inhibitory area being kept up. Then after a time the ventricles give a beat, and after a long diastolic interval another beat, and so on in slow rhythmic succession, the inhibitory area being still under stimulation. . . . Stimulation of the inhibitory area being now discontinued, the auricles as a rule soon come to a state of rest; then after a pause they beat in the ordinary fashion and lead off the ventricular action at pretty nearly the normal rhythm." According to MacWilliam the inhibitory area contains many nerve cells and ganglia and has numerous nervous connections establishing a close relation with both auricles and ventricles. The ventricular inhibition resulting from stimulation of the inhibitory area was considered to be a consequence of the excitation of certain inhibitory structures within it. A condition somewhat similar to that induced by experimental stimulation of the inhibitory area is observed in the human heart when the auricles are fluttering and the vagus is stimulated.

More than twenty years after MacWilliam first described auricular flutter, Hirschfelder[1] recorded a rapid rhythmic auricular action when faradic stimulation of moderate intensity was applied to the auricular appendix. Upon stronger stimulation there was no longer a co-ordinate contraction of the whole auricular musculature, but inco-ordinate fibrillar contraction.

The auricular contractions in flutter differ from those in fibrillation in being co-ordinate. There is, however, a form of auricular activity in which flutter and fibrillation are combined. This complex form of auricular activity was described by Rothberger and Winterberg[2] as "unreines Schlagen"—visible auricular contractions involving at least the main mass of the musculature together with either distinct fibrillary movements, especially at the margins of the auricles, or weak peristaltic waves. A similar condition in the dog's auricles was subsequently described by Canby Robinson[3] as resulting from stimulation of the right vagus after an auricular tachycardia had been induced by faradisation. Korteweg[4] has also described a form of auricular action

[1] Hirschfelder, A. D., "Contributions to the Study of Auricular Fibrillation, Paroxysmal Tachycardia, and the so-called Auriculo- (Atrio-) ventricular Extrasystoles," *Bull. Johns Hopkins Hosp.*, Baltimore, 1908, xix., 322.

[2] Rothberger, J., and Winterberg, H., "Ueber das Elektrokardiogramm bei Flimmern der Vorhöfe," *Arch. f. d. ges. Physiol.*, Bonn, 1910, cxxxi., 387.

[3] Robinson, G. Canby, "The Influence of the Vagus Nerves on the Faradized Auricles in the Dog's Heart," *Journ. of Exp. Med.*, New York, 1913, xvii., 429.

[4] Korteweg, A. J., *Arhythmie door Atrium-fibrillatie*, Proefschrift, Leiden, 1913.

intermediate between fibrillation and flutter as a result of vagus stimulation while the auricles were being faradised.

In a later paper by Canby Robinson [1] the tumultuous activity of the dog's auricles under faradisation is described as consisting of very rapid contractions of the whole auricles (flutter) and simultaneous fine fibrillatory movements. When the peripheral end of the right vagus was stimulated the general contractions ceased, while the fibrillation persisted. Moreover the activity of the faradised auricles of the dog might pass spontaneously into one closely resembling auricular flutter in man, and this change might also occur during stimulation of the left vagus.

The conclusions to which all these observations lead are—(1) the auricular activity which is induced by faradisation of the mammalian auricles is often a combination of flutter and fibrillation, but under certain conditions may be either flutter alone, similar to that in man, or fibrillation alone, as observed in the human auricles; (2) experimental flutter may be changed into fibrillation, or fibrillation into flutter; and (3) these two abnormal forms of auricular activity in the heart of man or of other mammals are closely allied disorders.

We know that auricular flutter in the human heart may persist, not merely for minutes or hours, but for months or even for years. The persistence of flutter, however, does not necessarily imply persistence of the exciting cause. In his original papers MacWilliam stated that flutter might continue for a "considerable time" after the stimulation had ceased. Moreover, according to Lovén,[2] Strömberg and Tigerstedt,[3] and Engelmann,[4] the application of a single induction shock to the great veins, the sinus, or the auricle of the frog's heart, sometimes evokes a series of accelerated rhythmic beats. The series starts abruptly, may last for two minutes, and terminates abruptly. The phenomenon is analogous to the series of rapid rhythmic ventricular contractions described by Gaskell [5] as following the application of a single stimulus to the auriculo-ventricular ring of muscle.

[1] Robinson, G. Canby, "The Relation of the Auricular Activity following Faradization of the Dog's Auricle to Abnormal Auricular Activity in Man," *Journ. of Exp. Med.*, New York, 1913, xviii., 704.

[2] Lovén, *Mitteilungen vom physiol. Laboratorium in Stockholm*, 1886, iv., 16.

[3] Strömberg and Tigerstedt, *ibid.*, 1888, v., 43.

[4] Engelmann, Th. W., "Ueber den Ursprung der Herzbewegungen und die physiologischen Eigenschaften der grossen Herznerven des Frosches," *Arch. f. d. ges. Physiol.*, Bonn, 1897, lxv., 109.

[5] Gaskell, W. H., "The Contraction of Cardiac Muscle," *Schäfer's Text-book of Physiology*, Edin. and Lond., 1900, ii., 179.

The close analogy between auricular flutter induced by faradic stimulation of the auricular appendix and auricular flutter in the human heart renders it probable that the latter likewise results from stimulation, excessive in frequency and intensity, of a localised area in some part of the auricular wall. We do not yet know how this effect is induced. A localised inflammatory area—an irritative lesion—in the sinus node, in the atrio-ventricular node, in either auricular appendix or in the general auricular musculature, might conceivably induce auricular flutter, but no well-defined case with a single focal lesion has yet been recorded. As tachycardia may be induced by the application of heat to the coronary sinus, after destruction of the sinus node, Zahn [1] has suggested that the primitive tissue in the vicinity of the coronary sinus may be the site of stimulus production in auricular flutter.

As the abolition of vagus control, by means of either atropin or bilateral section of the vagi, is accompanied by marked acceleration of the auricles, is it not possible that auricular flutter may be due solely to loss of vagus control? This hypothesis is improbable, because vagus stimulation, although retarding the ventricles when the auricles are in flutter, either converts the flutter into fibrillation or induces a combination of flutter and fibrillation, but does not retard the auricular beats. Again, as excessive stimulation of the accelerator nerves can excite auricular extrasystoles, may it not be an important factor in the production of auricular flutter also? Possibly this may be true in some instances when paroxysmal attacks of auricular flutter occur in a heart that is otherwise healthy; but when auricular flutter persists for months it is more likely to be due to other causes.

In the vast majority of cases of flutter there is almost certainly an irritative lesion in the auricular wall. But in some instances, and especially those in which emotion or other nervous influences induce attacks of auricular flutter, loss of vagus control and excessive accelerator stimulation may be concomitant factors.

Age.—Auricular flutter has been recognised in patients whose ages have ranged from 5 to 74 years. Analysing 49 cases in which the ages were stated, we find that two were under 10 years of age, four were between 10 and 19, three between 20 and 29, six between 30 and 39, eight between 40 and 49, eleven between 50 and 59, thirteen between 60 and 69, while two were over 70 years of age.

[1] Zahn, A., "Experimentelle Untersuchungen über Reizbildung und Reizleitung im Atrioventrikularknoten," *Arch. f. d. ges. Physiol.*, Bonn, 1913, cli., 247.

Sex.—About 85 per cent. of the recorded cases have been in males.

Arterial Disease.—The age incidence, as recorded above, indicates the probable relation between arterial degeneration and auricular flutter. In a number of cases the radial and brachial arteries are palpably thickened, and systolic pressures as high as 260 and 275 mm. Hg. have been recorded.

Other Evidence of Heart Disease.—In nearly all the cases there has been some evidence of cardiac enfeeblement. In some instances this has preceded, but in others it has followed, the attack of flutter. A definite history of antecedent acute rheumatism was elicited in only about 12 per cent. of cases; but about 30 per cent. have presented signs of mitral disease. In many of these cases an inflammatory process, starting in the mitral valve, had probably spread into the walls of the auricles. In other cases there was probably a more diffuse, sub-acute or chronic, myocarditis of unascertained origin.

Auricular flutter has been known to arise during the course of diphtheria (Cases XLIII. and XLIV.), influenza (Cases XXV. and XXXIV.), follicular tonsillitis, gastro-intestinal catarrh, and other acute infective diseases. It is not improbable that under such circumstances there were inflammatory foci in the auricular musculature. Flutter may arise in the late stages of exophthalmic goitre (Case VII.), and during chloroform anæsthesia (Case IX.). In the latter connection it is of interest to remember that this drug may provoke extra-systoles, either single or multiple, and also fibrillation of the auricles and ventricles (Levy,[1] Hecht and Nobel[2]). Of 49 cases, there was evidence of syphilis in five (Cases I., II., XXXVI., XXXVII., and XLIX.), and in two of these there was aortic incompetence.

[1] Levy, A. G., "The Exciting Causes of Ventricular Fibrillation in Animals under Chloroform Anæsthesia," *Heart*, Lond., 1912-13, iv., 319.

[2] Hecht, A. F., and Nobel, E., "Elektrokardiographische Studien über Narkose," *Zeitschr. f. d. ges. exper. Med.*, Berlin, 1913, i., 23.

CHAPTER III

MORBID ANATOMY

Although the clinical evidence indicates the existence of mitral disease in nearly one-third of the cases recorded up to the present time, comparatively little is known regarding the morbid anatomy of the heart in auricular flutter. It is evident, however, that if the disorder is due to a cardiac lesion, this must be sought for in the walls of the auricles. Moreover, the experimental and clinical evidence indicates that the pathological changes are probably of a nature similar to those in the hearts of individuals who, during life, had presented auricular extrasystoles or auricular fibrillation.

In Case XII., recorded by George A. Gibson,[1] the post-mortem examination revealed a chronic pericarditis which had led to obliteration of the sac. The heart was dilated and hypertrophied, but there was no valvular lesion. On microscopic examination, the walls of the auricles and ventricles were perfectly healthy, save for a slight increase of fibrous tissue close to the epicardium and obviously resulting from the pericarditis. The atrio-ventricular bundle was unduly fibrous; the vagus nerves were healthy; the lungs, liver, and kidneys presented advanced chronic venous congestion, and in the liver there was one small hard nodule resembling a calcified gumma.

In Case III. recent fibrinous pericarditis was found over the surface of the right auricle. All the chambers of the heart were dilated. The segments of the tricuspid valve were shrunken and thickened; the pulmonary valve was healthy. The mitral orifice was constricted, measuring only 3 cm. in circumference; the mitral cusps were shrunken, thickened, and partially calcified. The chordæ tendineæ also were thickened and shortened, while the papillary muscles were fibrous. To the naked eye no abnormality was visible in the pars membranacea septi. The aortic cusps were thick and shrunken, but not calcareous. The walls of the coronary arteries and their branches contained atheromatous patches, some of which were calcareous. The walls of the

[1] Gibson, G. A., "A Discussion on some Aspects of Heart-Block," *Brit. Med. Journ.* Lond., 1906, ii., 1113.

auricles and ventricles presented a slight degree of fibrosis throughout, while the lower part of the auricular septum was involved in a recent inflammatory process spreading into it from the anterior cusp of the mitral valve (Fig. 28).

The sinus node and the atrio-ventricular node and bundle were cut in serial section. The epicardium on the proximal and distal sides of the cavo-auricular junction presented a considerable infiltration with lymphocytes, together with a few plasma cells and an occasional polymorphonuclear leucocyte. This infiltration extended inwards

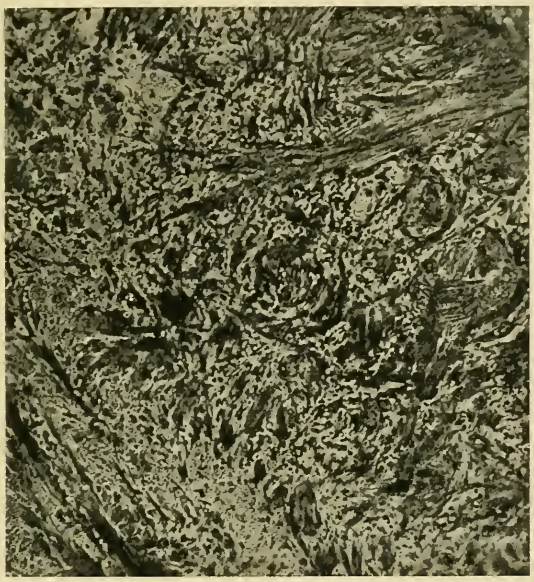


Fig. 28.—Inflammatory infiltration of the auricular musculature near the atrio-ventricular node. (×133.)

through the caval and auricular walls, and involved the sinus node and the numerous nerves and ganglia in its vicinity (Plate I., Fig. 2).

The atrio-ventricular node presented no abnormality except a slight lymphocytic infiltration, which was of lesser severity than that elsewhere in the auricular septum. The greater part of the main stem of the atrio-ventricular bundle was entirely free of inflammatory or degenerative changes. A sub-acute inflammatory process, however, extended inwards from the septal cusp of the tricuspid valve so as to involve the terminal portion of the bundle and, to a lesser extent, the initial portions of both its branches.

It was evident, therefore, that the various pathological changes in this heart were due to—(1) atheroma of the coronary arteries; (2) endo-

carditis spreading into the auricular septum from the mitral valve; (3) endocarditis extending inwards from the tricuspid valve so as to involve the atrio-ventricular bundle and its branches; and (4) acute pericarditis implicating the sinus node and the abundant nerve elements in its vicinity.

In Case VII. (exophthalmic goitre and myocarditis) the post-mortem examination was made by Dr. James Miller. The form of the heart was found to be similar to that outlined in an orthodiagram seventeen days before death. The maximum transverse diameter was 10·5 cm.; the long diameter from the superior cavo-auricular junction to the apex was 13 cm. The left auricle and its appendix, which had not been visible on the screen, were not exposed until the heart was rotated to the right on its long axis.

The pericardium was healthy. The jugular veins, the venæ cavæ, and the right auricle were dilated. The wall of the latter chamber was thin, and on microscopic examination marked diffuse inter-fascicular fibrosis was seen. In some of the pectinate muscles the muscle fibres had lost their striation, and the nuclei failed to take the stain, but there was very little infiltration with inflammatory cells. The tricuspid orifice measured 14·5 cm. in circumference; its valve was healthy. The cavity of the right ventricle was dilated, and its wall slightly hypertrophied. Sections taken from the conus arteriosus presented no abnormality except slight increase of connective tissue between the muscle fibres. The pulmonary orifice measured 9 cm. in circumference, and its valve was healthy.

The cavity of the left auricle was dilated and the endocardium thickened. Its wall was very thin, and on microscopic examination presented considerable diffuse inter-fascicular fibrosis; but no lymphocytic infiltration, and none of the degenerative changes observed in the wall of the right auricle, were detected. The mitral orifice measured 12 cm. in circumference; the valve segments were somewhat thick and fibrous. The muscle of the left ventricle was of dark colour and somewhat soft. Microscopic examination revealed small patches of fibrosis throughout the ventricular wall, but particularly in the apices of the papillary muscles and in the sub-endocardial layers of the columnæ carneæ. The aortic orifice measured 8 cm. in circumference; the basal portions of the cusps were thickened, but the valve was competent to the water test.

The pars membranacea septi was large, thin, and free from inflammatory reaction. The auricular septum in front of the mouth of the

coronary sinus was likewise healthy. The atrio-ventricular node, the bundle and the upper portions of both branches, were cut serially. The only abnormality was a very slight lymphocytic infiltration in some parts of the bundle and adjacent ventricular muscle. The atrio-ventricular node and both branches of the bundle were healthy.

The lungs and the abdominal viscera were markedly congested. The microscopic appearances of the enlarged thyroid gland were characteristic of exophthalmic goitre. The acini, of irregular shape, were lined by columnar cells and contained colloid material, while the amount of interstitial connective tissue was scanty.

In Case XI. the post-mortem examination was made by Dr. Murray Drennan. The heart was greatly enlarged, and all the chambers were dilated. The walls of both auricles were thin, and the right auricular appendix contained several adherent ante-mortem thrombi. The tricuspid orifice was dilated, measuring 15 cm. in circumference; the pulmonary orifice measured 8 cm., the mitral 12 cm., and the aortic 8 cm. in circumference. The wall of the left ventricle was slightly hypertrophied. There were only two aortic cusps; both were large, thickened, and had calcareous nodules on their aortic surfaces, but the valve was competent. The wall of the aorta and the coronary arteries showed nodular atheroma. There was resolving pneumonia in the lower and middle lobes of the right lung and in the lower lobe of the left lung; the liver and spleen showed chronic venous congestion; and the kidneys were cystic. Neither the vagi nor the cervical sympathetic presented any change throughout their entire course.

Microscopic examination revealed a very slight degree of fatty degeneration of the ventricular muscle, and a slight fibrosis which was more marked in the auricles than in the ventricles. An acute cellular infiltration was seen in the lowest part of the auricular septum posteriorly, but both nodes, the bundle, its branches, and the tissues at the mouth of the coronary sinus were healthy.

In Case XLIII.,[1] one of diphtheria with terminal auricular flutter, the musculature of the auricles and ventricles had undergone fatty degeneration, and in the walls of the ventricles there was a pronounced degree of interstitial myocarditis. The atrio-ventricular node and bundle were healthy, as were also the valves.

In Case XLIV.[1] auricular flutter supervened on the eleventh day of

[1] Hume, W. E., "A Polygraphic Study of Four Cases of Diphtheria with a Pathological Examination of Three Cases," *Heart*, Lond., 1913-14, v., 25.

an attack of diphtheria, two days before death (see p. 77). All the chambers of the heart were found to be dilated, but the valves were healthy; the myocardium was pale and friable; the right auricular appendix was filled with an ante-mortem thrombus. The sinus node was infiltrated with mononuclear cells of the formative type; the capillaries within the node were engorged, and hæmorrhages had taken place from them; the muscle fibres of the sinus node were in a state of granular degeneration. The atrio-ventricular node was normal except for slight engorgement. The bundle and its branches were perfectly normal; the auricular musculature presented slight fatty degeneration; the ventricular musculature presented intense fatty degeneration but no interstitial myocarditis.

In conclusion, it is evident that the number of post-mortem examinations is still too small to permit of any final conclusions regarding the condition of the heart in auricular flutter. The available evidence nevertheless demonstrates that flutter may be associated with widespead acute or sub-acute inflammation and degenerative changes in the auricular musculature, and that these changes may involve both nodes and the bundle. These changes may arise in the course of an acute infective disease. More frequently they are due to disease of the coronary vessels or to inflammation extending into the muscle from the pericardium or from the mitral and tricuspid valves.

CHAPTER IV

RECORDS OF CASES

Eleven cases that have come under my own observation will first be described, and thereafter other cases recorded in the literature will be referred to.

Case I.—Auricular Flutter with Complete Heart-block; in Two Initial Attacks of Flutter the Maximum Auricular Rates were 275·5 and 300 per Minute respectively; a Third Attack Persisted for nearly Five Years; between the Attacks the Auricles beat Slowly and Irregularly.[1]

The patient, a cabman, was aged 55 when seen for the first time in May 1904. His wife had two children, who died in infancy of diphtheria and pneumonia respectively, and three abortions. He denied syphilis, but when his blood serum was tested in 1909 it gave a positive Wassermann reaction.

When aged 16 he suffered from inflammation of the kidneys. At the age of 50 he became affected with muscular rheumatism, and eighteen months later with bronchitis and dyspnœa. In November 1903 he suffered from acute lobar pneumonia, and subsequently became more short of breath and less able for his work. He then began to experience attacks of faintness and giddiness on exertion two or three times a week. He said the attacks were "a sort of sudden blindness, and he had to hold on to a railing lest he should fall." Each attack

[1] Records of this case are contained in the following papers :—

Cowan, J., and Ritchie, W. T., "Coupled Rhythms of the Heart," *Quart. Journ. of Med.*, Oxford, 1910-11, iv., 55 (Case II., pp. 60, 61).

Gibson, G. A., "Bradycardia," *Edin. Med. Journ.*, Edin., 1905, (N. S.), xviii., 9 (Case III.).

Gibson, G. A., "A Discussion on some Aspects of Heart-block," *Brit. Med. Journ.*, Lond., 1906, ii., 1113.

Jolly, W. A., and Ritchie, W. T., "Auricular Flutter and Fibrillation," *Heart*, Lond., 1910-11, ii., 177.

Ritchie, W. T., "Complete Heart-block, with Dissociation of the Action of the Auricles and Ventricles," *Proc. Roy. Soc. of Edin.*, 1905, xxv., 1085.

Ritchie, W. T., "Further Observations on Auricular Flutter," *Quart. Journ. of Med.*, Oxford, 1913-14, vii., 1.

lasted as a rule for ten or fifteen minutes. He had never actually lost consciousness.

On examination he was a well-nourished man, somewhat cyanotic, but not dropsical. The walls of his radial and brachial arteries were moderately and diffusely thickened. The arterial pulse was of large volume and rhythmic at a rate of 31-36 beats per minute. The heart was greatly enlarged, as is evident from the orthodiagraphic record obtained on the 16th August 1911 (Fig. 29). The forcible cardiac impulse, which had the same rate and rhythm as the arterial pulse, could be seen and felt in the sixth intercostal space as far out as 12 cm. from the mid-sternal line. The position of the apical impulse was not

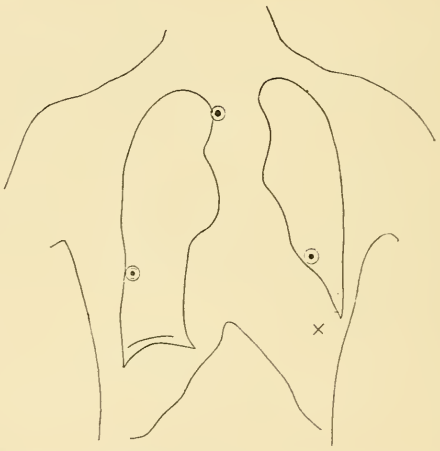


Fig. 29.—Orthodiagram of Case I. The heart is greatly enlarged and the aorta is prominent. The position of the supra-sternal notch, the nipples, and the apex-beat is indicated.

influenced by changes of posture; the epigastrium was indrawn with each ventricular systole, and Broadbent's sign was well marked. On auscultation a loud blowing systolic murmur of mitral origin was audible, but no other murmur could be heard, and no sounds were detected during the long diastolic pauses. The second sound, especially at the aortic area, was clear and accentuated. It was therefore evident that the patient was affected with mitral incompetence, chronic adhesive mediastino-pericarditis, and arterio-sclerosis. The lungs presented some vesicular emphysema and bronchitis, but examination of the abdomen, blood, urine, and nervous system revealed no abnormality.

The rate of the jugular pulsations was observed to be more

rapid than that of the arterial pulse. Complete dissociation of the ventricular rhythm from that of the auricles was first demonstrated in polygraph tracings taken by Dr. Oliphant Nicholson in 1904. Some of these tracings were reproduced in Dr. G. A. Gibson's paper in 1905. In other tracings that I obtained by means of the Knoll-Hering polygraph during the period from 26th March until 16th June 1905, complete heart-block was always demonstrated, the rates of the auricles and ventricles being 50·7-70·5 and 31·1-35·7 per minute respectively

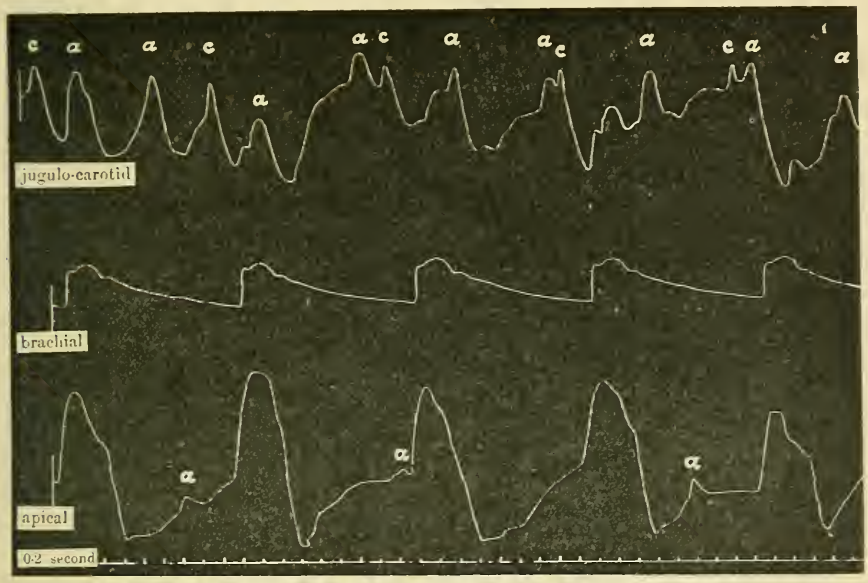


Fig. 30.—Complete heart-block. Jugulo-carotid, brachial, and apical tracings. The time record is 0·2 second. (Case I. before the onset of auricular flutter.)

(Fig. 30). The patient's history was uneventful until, on the 2nd June 1905, while cleaning a window, he had a "giddy attack" and fell from a ladder. He did not hurt himself. This was the last manifestation of the Adams-Stokes syndrome. On the following day his ventricular rate was 40 per minute after he had walked a distance of one mile, and 34·75 per minute after resting for two hours. On the 16th June the auricles were beating somewhat irregularly at a rate of 60·71 per minute, while the ventricular beats were rhythmic at a rate of 32 per minute. On the 19th June 1905, when the patient's heart was examined by radioscopy, his right auricle was observed to be beating rhythmically about 60 times per minute, and to have a rhythm independent of the slower ventricular beats.

TABLE I.

	Rate per Minute.		
	Auricles.	Ventricles.	
1905. March 26	64·78	33·80	Both auricles and ventricles are beating rhythmically.
April 16	70·5	32·9	
„ 30	66·8	35·7	
May 19	50.76	32·30	Auricles slightly arrhythmic.
June 2	...	...	Syncopal attack. Fell from ladder.
„ 9	65·8	31·14	Auricles somewhat arrhythmic.
„ 16	60·71	32	
„ 19	...	...	Examined by Röntgen rays. Auricles beating 60 times per minute.
„ 20	...	...	Patient began to take $\frac{1}{100}$ grain of atropin sulphate thrice daily.
„ 23	273·03	34·88	
	274·73	36·58	Ten minutes after $\frac{1}{50}$ grain of atropin sulphate.
„ 27	...	...	Atropin stopped.
„ 30	275·5	30·0	Both auricles and ventricles are beating rhythmically.
July 8	270·77	30·77	
October 14	43·70	31·57	
1906. June 3	290·90	32·96	
	300·0	33·80	
1908. May 28	57·39	31·30	Ventricles are arrhythmic.
August 25	53·33	33·60	Both auricles and ventricles are somewhat arrhythmic.
November 19	291·89	33·61	Both auricles and ventricles are rhythmic.
„ 19	272·72	33·33	
„ 19	267·85	34·09	
„ 21	272·72	33·33	
1909. February 22	268·75	35·29	A few ventricular extrasystoles.
March 8	255·55	42·85	Both auricles and ventricles are rhythmic.
July 28	260·86	31·91	
August 19	262·5	33·3	After bilateral pressure on the vagi.
„ 19	257·0	34·2	After exercise.
„ 20	234·63	31·91	Both auricles and ventricles are rhythmic.
„ 25		56	Group-beating of ventricles.
„ 26	252·63	34·42	A few ventricular extrasystoles.
September 20	276·9	32·4	Both auricles and ventricles are beating rhythmically.
„ 30	261·11	56·7	Group-beating of ventricles.
October 4	260·86	58·9	
„ 9	268·1	41·02	10.30 A.M. Ventricular arrhythmia.
„ 9	...	41·74	10.40 A.M.
„ 9	...	...	10.51 A.M. $\frac{1}{50}$ grain of atropin sulphate subcutaneously.
„ 9	263·8	...	10.56 A.M.
„ 9	...	44·21	10.57 A.M.
„ 9	...	44·6	11.3 A.M.
„ 9	268·6	44·44	11.11 A.M.
„ 9	...	48·2	11.12 A.M.

	Rate per Minute.		
	Auricles.	Ventricles.	
1909. October 9	...	45·2	11.19 A.M.
„ 9	260·8	44·0	11.27 A.M.
„ 14	272·7	51·5	Group-beating of ventricles. Has been taking digitalis for five days.
November 13	...	...	Ventricles beating rhythmically.
„ 22	251·18	63·15	Group-beating of ventricles.
„ 29	...	...	Digitalis stopped.
December 2	250·0	34·0	Ventricular extrasystoles.
1910. January 13	265·55	37·50	Ventricular arrhythmia.
February 3	280·03	35·55	Ventricles more rhythmic.
„ 10	252·99	31·92	Ventricles rhythmic.
March 24	258·46	32·43	Occasional ventricular extrasystoles.
„ 24	246·77	32·70	
April 28	254·23	36·20	Ventricles rhythmic.
May 16	264·5	35·5	
August 8	251·35	34·61	Ventricles rhythmic.
September 16	250·0	55·3	Group-beating of the ventricles.
October 10	253·8	60·5	„ „ „
November 16	250·0	55·3	„ „ „
December 27	250·0	50·5	Neither right nor left vagus compression retards auricles or ventricles.
1911. February 8	250·0	62·5	Group-beating of the ventricles.
April 24	...	36·5	Ventricles rhythmic.
„ 27	252·6	41·5	Ventricles rhythmic. Before inhalation of amyl nitrite.
„ 27	256·4	41·4	After inhaling amyl nitrite for four minutes.
May 11	...	45	Maximum systolic pressure, 165 ; diastolic pressure, 125 mm. Hg.
July 10	277·7	32·4	Respiratory rate the same as the ventricular.
July 17	267·0	33·5	Ventricles rhythmic.
August 18	242·8	37·9	„ „
October 26	232·7	33·9	„ „
November 1	232·4	33·6	„ „
„ 15	229·5	32·0	Ventricles rhythmic. Has been taking 30 minims of tincture of squill daily since 19th October.
December 8	244·9	37·4	Ventricles rhythmic.
1912. January 4	244·9	52·4	Ventricles arrhythmic.
„ 8	244·9	39·3	Ventricular beats often coupled.
August 22	244·8	55·7	Plate IX., Fig. 36.
September 25	244·4	38·2	Plate IX., Fig. 38.
October 22	242·5	38·0	Ventricles arrhythmic.
December 16	235·7	32·7	Ventricles rhythmic except for an occasional premature beat.
1913. March 10	190·5	44·8	Group-beating of the ventricles.
April 11	194·8	47	Numerous premature ventricular beats.
„ 24	194·8	35·7	Ventricles rhythmic.
May 2	201·0	34·9	„ „
„ 9	192·6	42·4	Group-beating of the ventricles.
„ 13	187·8	31·7	Ventricles rhythmic.
„ 19	192·8	33·3	2.32 P.M.
„ 19	189·0	40·3	2.35 P.M.
„ 19	189·4	42·4	2.40 P.M.

		Rate per Minute.		
		Auricles.	Ventricles.	
1913. May	19	...	...	2.42 P.M. 0·03 grain atropin sulphate subcutaneously.
„	19	192·8	37·9	2.52 P.M.
„	19	193·5	40·6	3.2 P.M.
„	19	195·9	41·4	3.8 P.M.
„	19	197·8	42·0	3.14 P.M.
„	21	190·5	31·3	11.0 A.M.
				11.5 A.M. 0·001 gramme strophanthin (Boehringer) intravenously.
„	21	92·1	29·0	1.35 P.M.
„	21	119·6	41·0	1.40 P.M.
July	15	201·6	40·6	An occasional ventricular extrasystole.
„	22	247·8	51·4	Ventricles arrhythmic and extrasystoles.
„	29	235·7	39·8	Ventricles rhythmic.
August	15	237·9	38·1	An occasional ventricular extrasystole.
„	29	240·0	38	Admitted to hospital with congestion at base of left lung.
„	30	236·8	41	
„	31	233·1	43	Began to take digitalis tincture. Ventricles irregular.
September	1	232·2	38·3	
„	2	230.7	34	Ventricles almost wholly rhythmic.
„	3	233·7	34·6	Ventricles rhythmic.
„	4	240·0	34·2	
„	5	230·7	31·9	Digitalis stopped. Has taken 180 minims.
„	11	57·1	32·9	
„	19	...	...	Left hospital, much improved.
„	22	63·6	33·3	
October	2	244·9	36·8	
„	14	69·5	38·0	Both auricles and ventricles irregular.
„	15	45·6	41·4	„ „ „
„	17	52·2	42·3	„ „ „
„	21	71·4	38·4	„ „ „
„	31	58·7	36·3	„ „ „
November	21	236·0	38	Auricular deflexions are rhythmic and diphasic.
„	28	225·5	37·2	Auricular deflexions are rhythmic and diphasic.
December	4	227·5	32·6	
„	8	237	35·3	Ventricles rhythmic.
„	10	223·2	38	Ventricles somewhat arrhythmic.
1914. January	14	232·9	48·7	„ „ „
„	23	232·4	39·8	Ventricles rhythmic.

Four days later (23rd June 1905) the auricles were fluttering. The patient had been taking $\frac{1}{100}$ grain of atropin sulphate thrice daily for three days. He felt better, and was less breathless than formerly, but for the first time the rate of his auricles was observed to be greatly accelerated, the beats being rhythmic at a rate of 273·03 per minute, while the ventricular rate was 34·88. Ten minutes after

$\frac{1}{30}$ grain of atropin sulphate had been given subcutaneously the auricular and ventricular rates were 274·73 and 36·58 per minute respectively. He continued to take $\frac{1}{100}$ grain of atropin sulphate thrice daily until the 27th June, when he began to take 15 minims of strophanthus tincture daily. On the 30th June, after 55 minims of this tincture had been taken, he said he felt "all right"; the auricular and ventricular rates were 275·5 and 30·0 per minute respectively. On the 8th July 1905, a week after he had ceased taking strophanthus, he felt well. The cyanosis had disappeared. The auricular and ventricular rates were 270·77 and 30·77 per minute respectively.

He felt so well that he was able to follow his occupation as a coachman in a country hotel, driving carriages, grooming horses, and cleaning the stalls and harness. Whether his auricles were still fluttering, I cannot say. But after being thus engaged for a month, he began to be troubled with vomiting, which was probably induced by alcoholic excess. When he returned to town, feeling weak and troubled with cough, but neither cyanotic nor dropsical, his ventricles were beating rhythmically at a rate of 34·75 per minute. On the next occasion when records of his auricular action were obtained, namely, on the 14th October 1905, the auricular flutter had disappeared and had been replaced by an infrequent auricular rate of 43·70, while the ventricular rate was 31·57 per minute. During the following winter the patient felt well and able for any work he could have obtained. His ventricular rate was not observed to rise above 37 nor to fall below 32·5 per minute.

On the 3rd June 1906, without the patient having experienced any particular subjective phenomena, the auricular flutter was found to have recurred. The auricular rate was then 290·9-300 per minute. In May and August 1908 the auricular rate had again fallen to 53-57 per minute. On the 19th November 1908, however, the auricles were again fluttering at a rate of 267·8-291·8 per minute. In all the records taken from that day until July 1911, the auricular rate was usually between 250 and 275 per minute, and only once fell as low as 234·6. During the succeeding period of sixteen months (August 1911 until December 1912) the recorded auricular rates never rose above 244·9 nor fell below 229 per minute. Thus on every occasion during a period of four years, the flutter was recorded (Table I.). Subsequently the auricular rate was usually about 189-195 per minute. Still later, short periods of flutter at a rate of 230-244 alternated with others during which the auricular rate was infrequent (45-71 per minute).

During the prolonged attack of flutter, the patient was somewhat breathless and cyanotic from time to time, and the lower lobe of the left lung shewed transient congestion and œdema. The urine eventually contained a constant trace of albumin with hyaline and granular tube-casts. There has never been any œdema of the limbs, and the patient is still able to walk to hospital, a distance of about two miles from his home, without becoming unduly fatigued or breathless.

Polygraph Tracings.—When the auricles were fluttering, faint yet distinct rapid pulsatile movements could usually be seen in the jugular veins if the patient were in the recumbent posture. These

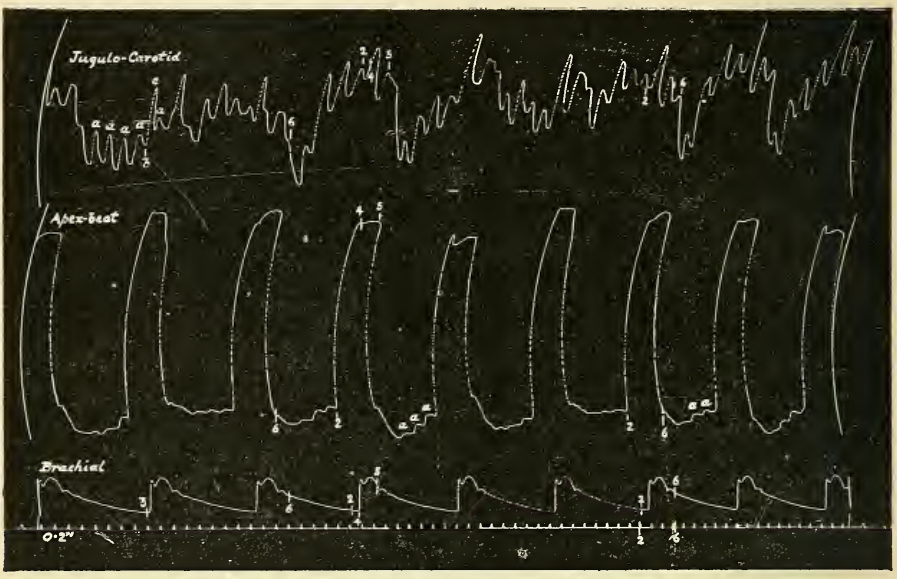


Fig. 31.—Auricular flutter and complete heart-block. The jugulo-carotid and apical tracings present a series of rhythmic, positive waves, the rate of which is about 255 per minute. The ventricles contract rhythmically 32·2 times per minute. *Heart*, vol. ii.)

movements were recorded graphically on many occasions. Fig. 31 shows the jugulo-carotid, apical and brachial tracings recorded simultaneously from this case in 1905. The rhythmic beats of the auricles at a rate of 255 per minute are represented by rhythmic waves, *a*, in the venous and apical tracings. The ventricles were contracting rhythmically at a rate of 32·2 per minute.

Electrocardiograms.—The first electrocardiograms from this patient were recorded by Jolly in 1909. He demonstrated that the rhythmic auricular deflexions (Fig. 32) were brought to light more clearly when the tension of the fibre was reduced until its deflexion time was 0·031 second than when its tension was greater and its deflexion time was 0·022 second. He also proved that these rhythmic auricular deflexions

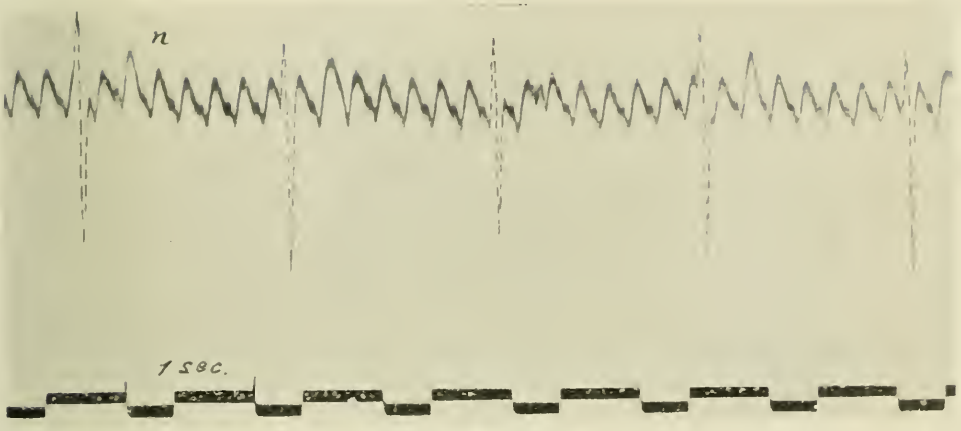


FIG. 32.—Auricular flutter, at a rate of 276 per minute, and complete heart-block. Case I. Derivation III. (From *Heart*, vol. ii.)

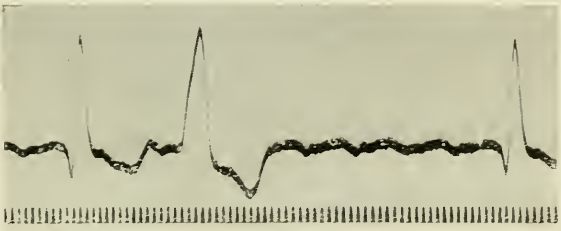


FIG. 33.—Auricular flutter, at a rate of 245 per minute, and complete heart-block. The second ventricular beat is an extrasystole. Case I. (21st July 1913). Derivation I. 1·5 cm. = 1 millivolt.

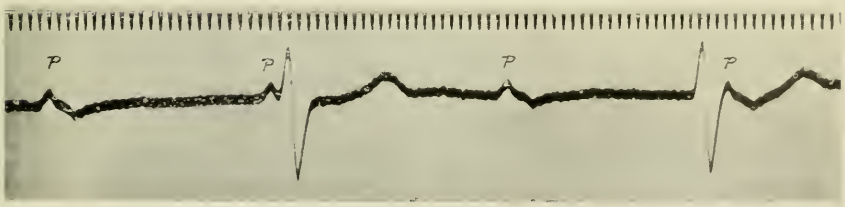


FIG. 34.—Case I. The flutter has ceased and the auricles are beating at a rate of 65·5 per minute. The auricular deflexions are diphasic. The block is still complete. Derivation II., 14th October 1913. 1·5 cm. = 1 millivolt.

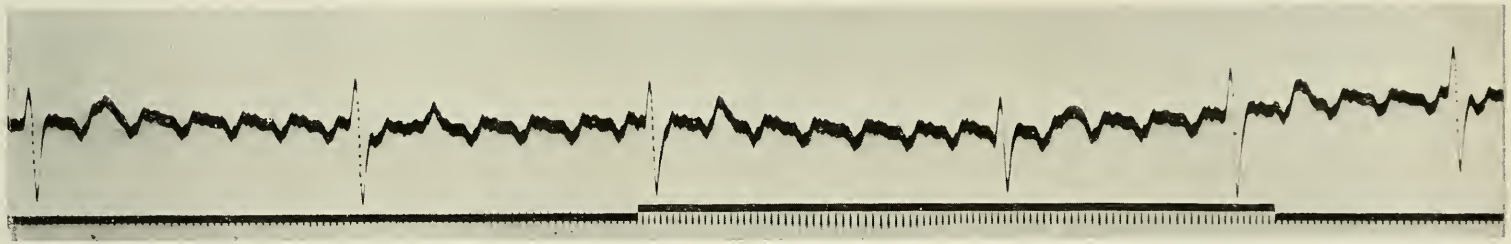


FIG. 35.—Auricular flutter, at a rate of 244 per minute, and complete heart-block. The signal indicates the period of compression of the left vagus. This does not retard either auricles or ventricles. Case I. Derivation II. 1·5 cm. = 1 millivolt.

PLATE IX.

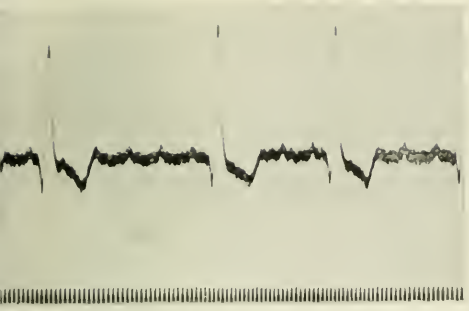


Fig. 36.

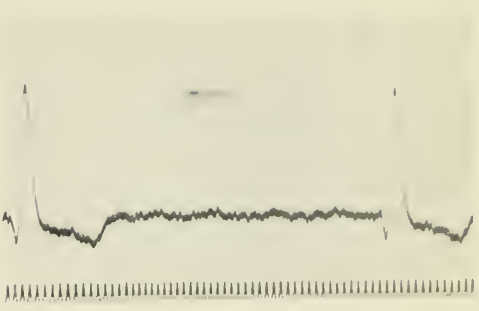


Fig. 37.

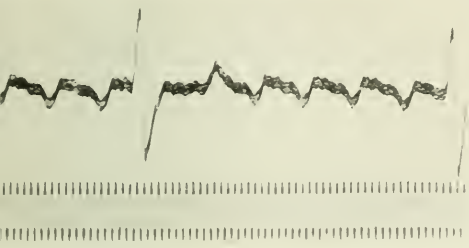


Fig. 38.

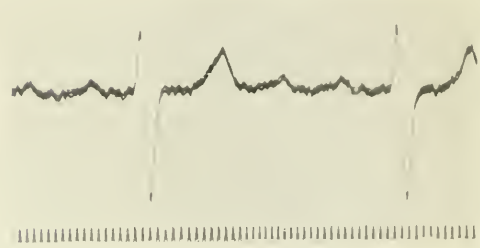


Fig. 39.

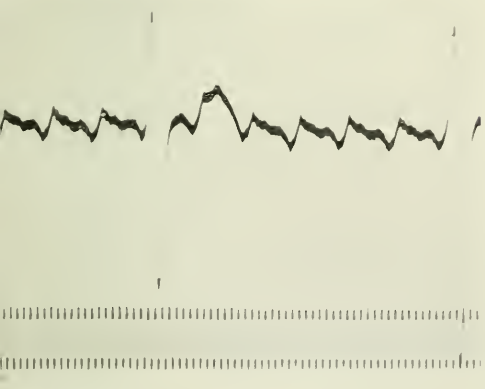


Fig. 40.

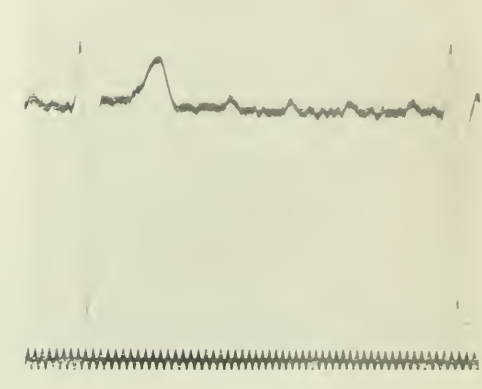


Fig. 41.

Figs. 36, 38, 40.—Auricular flutter and complete heart-block. Case I. Derivations I. (22nd August 1912), II. (25th September 1912), and III. (25th September 1912) respectively. The auricular rate is 244·8, 244·4, and 244·4 per minute, and the total value of the auricular deflexions about 70, 200, and 200 microvolts respectively. In Figs. 38 and 40 the auricular deflexions are diphasic. 1·5 cm. = 1 millivolt.

Figs. 37, 39, 41.—Case I. Derivations I., II., and III. respectively. In Fig. 37 the auricular deflexions are inconspicuous. In Figs. 39 and 41 the auricular rates are 194·8 and 197·7 per minute respectively, and the value of the auricular deflexions is about 100 microvolts (11th April 1913). 1·5 cm. = 1 millivolt. (*Quart. Journ. Med.*, vol. vii.)

did not represent tremor of skeletal muscles, ventricular beats, contractions of caval musculature at a greater rate than that of the auricles, or fibrillar contraction of the auricular musculature. The deflexions were recognised, therefore, to be rhythmic co-ordinate contractions of the auricular musculature. By derivation I. each auricular contraction was represented by a simple upward deflexion. By derivations II. and III. each auricular deflexion was diphasic, being composed of an initial deflexion upwards succeeded after an interval, during which the shadow of the fibre traced a horizontal line, by a downward deflexion. The upward deflexion was less acute than the downward. The duration of the former was about 0·1 second, that of the latter 0·059 second. As recorded by derivation I., the auricular deflexions were shown to have a value of about 60 microvolts, as compared with 100-200 microvolts in the case of the auricular deflexions of a healthy human heart.

In all the subsequent electrocardiograms until December 1912, the form of the auricular deflexions as recorded by each derivation was constant. Derivation I. gave a simple rise and fall, with a value of 60-70 microvolts. Derivations II., III., and IV. yielded diphasic curves with an initial upward deflexion and a total value of about 200 microvolts (Figs. 36, 38, 40). Atropin did not accelerate the auricular rate, and compression of the right or left vagus in the neck did not arrest, or even retard, the auricles (Plate VIII., Fig. 35).

During a period of about three months in the spring of 1913 the auricular rate was as a rule only about 190-194 per minute, with a maximum of 201 per minute. Subsequently a more frequent rate (223-247) was again maintained. In contrast with the antecedent and subsequent auricular flutter, the less frequent auricular action was represented by deflexions of lesser amplitude, and by derivations II. and III. they were no longer uniformly diphasic (Figs. 37, 39, 41). Further, the auricular action could then be accelerated by means of atropin and retarded by means of strophanthin administered intravenously. It is probable that the true auricular flutter, which was not under vagus control, and the less frequent auricular tachysystole, were not initiated at the same site in the auricular wall. The evidence at present available suggests that the chronotropic influence of the vagus upon the auricles is exerted mainly through the sinus node, and consequently the site at which the slower auricular rhythm was initiated was probably nearer the sinus node than that of the true flutter.

When the auricles, in October 1913, again beat slowly and irregularly their electrocardiographic deflexions were of more normal form than when the auricles were in flutter (Plate VII., Fig. 34).

The Ventricles.—For five years after the heart-block was recognised the ventricular rate was constantly about 32-36 per minute, and the rhythm was absolutely regular. In March 1909 a few ventricular extrasystoles were recorded. They were either interpolated, or followed by a diastolic pause as long as that after each rhythmic beat. Four years later, electrocardiographic records of ventricular extrasystoles were obtained. By derivations I. and II. their initial deflexion was upwards (Plate VII., Fig. 33).

Another form of ventricular irregularity was more frequently encountered. It consisted in group-beating of the ventricles with a

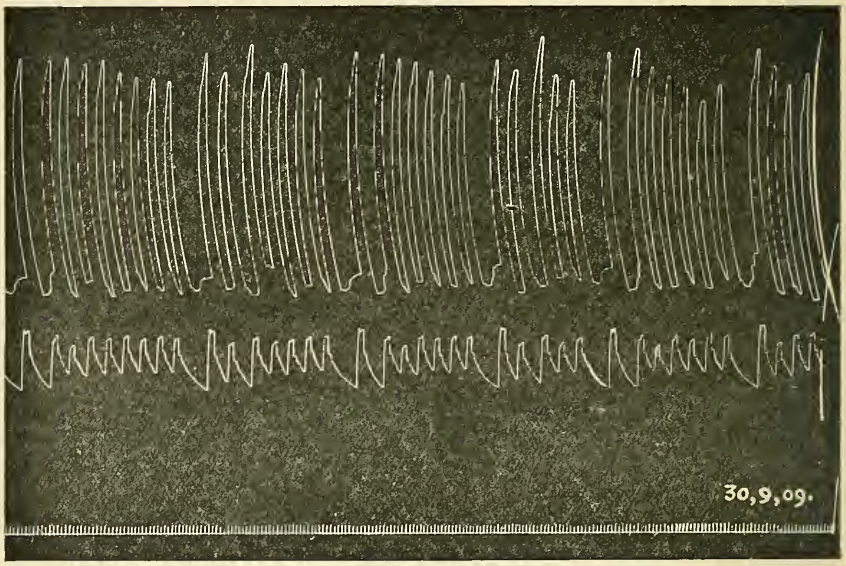


Fig. 42.—Apical and brachial tracings from Case I., showing group-beating of the ventricles. The time record is 0·2 second.

rate of 41-63 per minute. There were groups of two to eleven, and sometimes about twenty, rapid ventricular beats (Fig. 42), each group terminating in a long diastolic pause of about the same duration as that after a systole when the ventricles were beating rhythmically and less frequently. The following figures represent inter-ventricular periods in seconds and show the group-beating:—

1·80, 1·00, 0·90, 0·94, 0·86, 0·92, 0·90, 0·97, 0·92, 0·90, 1·76
1·68, 1·40, 1·00, 0·88, 0·90, 0·92, 0·88, 0·92, 0·92, 0·92, 0·90 . . .
1·76, 1·44, 1·00, 0·92, 0·96, 0·90, 0·95, 0·90, 0·93, 0·96, 0·95, 1·72
1·76, 1·44, 0·96, 0·95, 0·95, 1·80
. . . 0·94, 0·90, 0·94, 0·90, 0·90, 1·84
1·86, 1·00, 1·00, 1·24, 1·00, 1·00, 1·24, 1·04, 1·00, 1·30, 1·84.

The diastolic pause after the first systole of a group was often longer than that after the second; this was usually longer than that after the third and subsequent systoles. There was no lessening of the frequency of the ventricular rate towards the end of a group comparable to that recorded by Wenckebach.[1] The jugulo-carotid tracings demonstrated that while the ventricles were in group-beating the auricles were in constant, rapid, rhythmic flutter at a rate of about 276·9 per minute.

The group-beating of the ventricles was recorded electrocardiographically on many occasions. The ventricular deflexions were not atypical but on the contrary were of the same form as those of the rhythmic series of beats at a rate of 32-36 per minute. The stimuli for all the ventricular beats, therefore, were initiated at the same site, namely, in the main stem of the atrio-ventricular bundle below the level of the severing lesion. The group-beating of the ventricles in this case is evidently analogous to the series of rapid rhythmic ventricular beats described by Gaskell[2] as following the application of a single stimulus to the auriculo-ventricular ring of muscle, and likewise to the group-beating of the ventricles after the experimental production of heart-block as recorded by Erlanger and Blackman.[3]

Case II.—Auricular Flutter, at a Rate of 268·4-283 per Minute, which Subsequently Passed into Fibrillation.[4]

A traveller's porter, aged 45 years, was admitted to the Royal Infirmary on the 9th August 1912 complaining of shortness of breath and of pain in the left side of the chest for two months. Twenty-four years previously he had suffered from syphilis, for which he was treated for three months. Following the primary sore his throat became affected, and most of his hair fell out, but he considered that he had been free of any subsequent ill effects. Four and a half years ago he was confined to bed for two months on account of rheumatic pains in the feet and legs; and two years ago he met with an accident, necessitating an amputation through the left forearm. He had been

[1] Wenckebach, K. F., "Beiträge zur Kenntnis der menschlichen Herztätigkeit," *Arch. f. Anat. u. Physiol.* (Physiol. Abt.), Leipz., 1908, Suppl., 53.

[2] Gaskell, W. H., "The Contraction of Cardiac Muscle," *Schäfer's Text-book of Physiology*, Edin. and Lond., 1900, ii., 179.

[3] Erlanger, J., and Blackman, J. R., "Further Studies in the Physiology of Heart-block in Mammals. Chronic Auriculo-ventricular Heart-block in the Dog," *Heart*, Lond., 1909-10, i., 177.

[4] "Auricular Flutter," *Edin. Med. Journ.*, Edin., 1912 (N.S.), ix., 485.

a moderately temperate man. With the exception of slight palpitation during the past twelve months, he was in good health until eight weeks ago, when he began to feel short of breath on carrying his parcels up flights of stairs. Day by day the dyspnœa became more pronounced, and he then began to suffer from pain in the left side of the chest and in the epigastric region. The pain usually came on at night, for example, when he was going upstairs after his day's work was done. The pain in the epigastrium was localised at an area three inches below the infra-sternal notch, and this area was tender on deep pressure. This pain bore no relation to the taking of food, but was always aggravated when the breathing was embarrassed, and became less severe when he sat quietly. He did not complain of nocturnal dyspnœa, he had never felt faint or lost consciousness, and he had never noticed any signs of dropsy.

On admission to hospital he was a well-nourished man, measuring 5 feet $8\frac{1}{2}$ inches, and weighing 133 lb. He was rather bald for his years, but presented no cyanosis or dropsy. The chest was well formed. The cardiac impulse was strong and unduly diffuse, the apex-beat being at a point in the sixth left intercostal space, four inches to the left of the mid-sternal line. As ascertained by percussion, the right border of the heart lay $1\frac{1}{2}$ inches to the right of the mid-sternal line. Over the mitral area a loud blowing systolic murmur was audible. It was conducted round the left side of the chest, and was faintly audible at the lower end of the sternum. A moderate degree of venous pulsation was visible on the right side of the neck. The arterial pulse was at the rate of 138 per minute, rhythmic, and of good force. The walls of the radial arteries were somewhat thick, but the vessels were not tortuous. The systolic blood-pressure was 152 mm. Hg, as ascertained by the Riva-Rocci sphygmomanometer. He had a slight cough, but no expectoration. The respiratory rate was 28 per minute. The breath sounds were of normal character, with a few medium crepitations and rhonchi at the base of each lung. Neither the liver nor the spleen was enlarged. The urine was somewhat scanty, had a specific gravity of 1024, and contained a trace of albumin. No tube casts were detected.

Progress.—16*th August.*—The patient has been resting quietly in bed, and taking 10 minims of tincture of digitalis with half a grain of sodium nitrite thrice daily since his admission, and now feels decidedly better. He passed 70 ounces of urine to-day. He still has palpitation, but no pain. The pulse has been constantly frequent—120-140 per minute—and rhythmic. The liver is now considerably enlarged,

extending down to the level of the umbilicus in the right mammillary line. Electrocardiograms recorded to-day show that the auricles are beating rhythmically at a rate of 283·0, while the rate of the rhythmic ventricular beats is exactly one-half, namely, 141·5 per minute (Plate X., Fig. 46). Pressure on either the right or on the left vagus

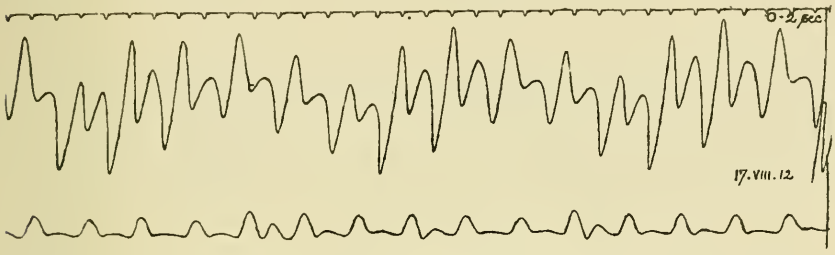


Fig. 43.—Auricular flutter with a ventricular response to every second auricular beat. The rate of the arterial pulse is 136·3 per minute. Jugulo-carotid and brachial tracings.

causes transient slowing of the ventricles, but they soon escape from vagus inhibition and resume their former rate of contraction.

17*th August*.—Polygraphic records show a ventricular venous pulse. The ventricles usually contract rhythmically at a rate of 136·3 per minute, each pulse period in the sphygmogram being about 0·44 second (Fig. 43). In other parts of the record there are groups of three to five rhythmic ventricular beats, each inter-systolic period measuring about

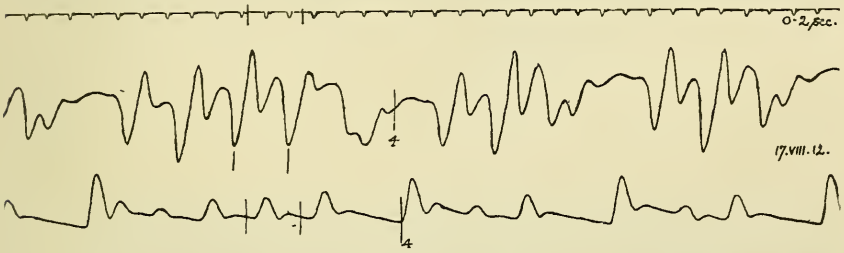


Fig. 44.—Auricular flutter, Case II. Jugulo-carotid and brachial tracings. The ventricles usually respond to every second, but occasionally to every third, auricular beat. Grouping and alternation of the arterial pulse-beats.

0·44 sec. Each group of beats is separated from that which precedes or that which follows it by a period of about 0·72-0·80 second, and the sphygmogram shows marked alternation of the pulse-beats in each group. The first beat of each group is represented by a large pulse-wave; the second ventricular beat is rhythmic, but the corresponding pulse-wave is small, and delayed in transmission (Figs. 44 and 45).

18*th August*.—The patient slept ten hours last night, and felt much better. In electrocardiograms the auricular deflexions occurred

rhythmically at a rate of 280·4 per minute. The ventricles were usually beating rhythmically at a rate of 140·2 per minute, in response to every second auricular stimulus, but occasionally there was a response to every fourth auricular beat. Pressure upon the right vagus caused transient slowing of the ventricles, while the auricular rate remained unchanged. The dose of digitalis tincture was increased to 20 minims thrice daily.

19*th August.*—The auricles were beating rhythmically at a rate of 276·4 per minute. The ventricular beats were usually rhythmic at a rate of 138·2 per minute—As : Vs : : 2 : 1; but every now and again the ratio was 4 : 1, and at these times the auricular deflexions were diphasic.

20*th August.*—The patient passed 95 ounces of urine, having a specific gravity of 1015, and still containing a small amount of albumin.

22*nd August.*—He slept about ten hours last night, has no dyspnœa

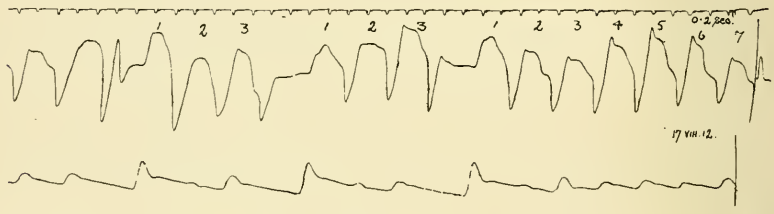


Fig. 45, Case II.—Tracings from the apex-beat and brachial artery, showing two groups, each consisting of three rhythmic ventricular beats, and seven rhythmic beats of another group. The groups are separated one from another by comparatively long diastolic pauses. The first ventricular contraction of each group is represented by a large wave in the sphygmogram; the second ventricular systole is represented by a small wave, which is delayed in transmission.

and no dropsy, and considers that he would be fit for his work if he were allowed to get up. The sodium nitrite was stopped, and the dose of digitalis tincture was reduced from 20 to 10 minims thrice daily. The auricles were beating rhythmically 276·4 times per minute. The ventricles usually responded to every fourth auricular beat, but the ventricular rhythm was sometimes irregular, there being a response to every third or fourth auricular stimulus (Plate XV., Fig. 82).

23*rd August.*—The radial pulse was rhythmic at a rate of 72 per minute.

24*th August.*—The arterial pulse was 68 per minute and rhythmic.

25*th August.*—The pulse was rhythmic at a rate of 72 per minute.

26*th August.*—The auricles were beating rhythmically at a rate of 268·4; the ventricular beats were rhythmic at a rate of 67·1 per minute—a constant ratio of As : Vs : : 4 : 1. The auricular deflexions were diphasic.

27*th August.*—Patient passed 70 ounces of urine, which was free of

PLATE X.

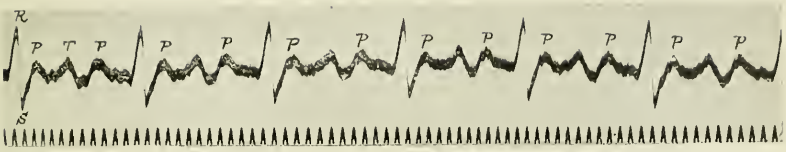


Fig. 46.—Auricular flutter with a ventricular response to every second auricular beat. Case II. Derivation II. 16, viii. 1912 (see p. 47).

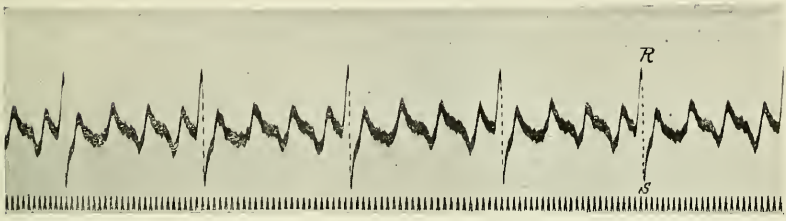


Fig. 47.—Auricular flutter with a ventricular response to every fourth auricular beat. Case II. Derivation II. 29, viii. 1912 (see p. 49).

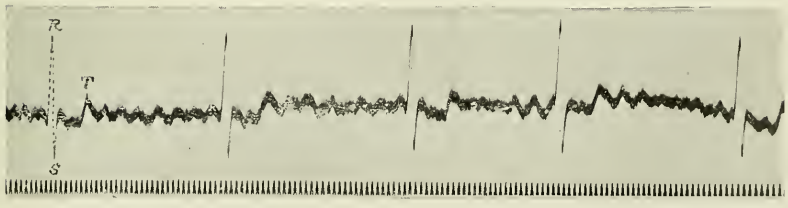


Fig. 48.—Auricular fibrillation. Case II. Derivation II. 9, ix. 1912. (*Edin. Med. Journ.*, N.S., vol. ix.)

albumin. The dose of digitalis was increased to 15 minims thrice daily.

28*th August.*—The auricles and ventricles were beating rhythmically at 274·28 and 68·57 per minute respectively, the ratio of As : Vs : : 4 : 1 being constant. The auricular deflexions were still diphasic. Pressure upon the right vagus did not retard the auricles, but caused transient slowing of the ventricles (Plate XVIII.). For each pulse-beat in the sphygmogram there were four waves in the jugulo-carotid tracing. The two waves occurring during ventricular diastole were small. The two waves occurring during ventricular systole were larger, and the first of them occurred immediately after, and was fused with, the carotid wave (Fig. 49).

29*th August.*—The auricles and ventricles were beating at rates of 281·0 and 70·25 respectively, with a constant ratio of As : Vs : : 4 : 1.

Fig. 49, Case II.—Jugulo-carotid and brachial tracings recorded on the 28th August 1912. The auricular and ventricular rates are 266·4 and 66·6 respectively.

30*th August.*—The dose of tincture of digitalis was increased to 20 minims thrice daily.

31*st August.*—The auricles and ventricles were beating rhythmically at rates of 274·28 and 68·57 respectively, with a constant ratio of 4 : 1, and the auricular deflexions were still diphasic.

2*nd September.*—The patient was allowed up yesterday; he slept for nearly ten hours last night, and had no pain or palpitation. To-day he was walking about the ward, and felt quite well. The auricles and ventricles were beating rhythmically at rates of 274·2 and 68·55 respectively, the ratio of As : Vs : : 4 : 1 being constant except when pressure upon the right vagus retarded the ventricles, although it exerted no influence on the auricular rate.

5*th September.*—The arterial pulse has remained uniformly rhythmic and its rate has varied only from 68 to 72 per minute. To-day the auricular rate is 274·2, the ventricular 68·55 per minute.

7*th September.*—Electrocardiograms taken yesterday showed that the ratio of As : Vs : : 4 : 1 was still constant. To-day the dose of digitalis tincture was increased to 30 minims thrice daily.

9th September.—The patient felt well, and was walking about the ward and corridor. The arterial pulse was found to be wholly irregular at a rate of 62 per minute. Electrocardiograms (Fig. 48) revealed auricular fibrillation. The auricular deflexions were at a rate of about 467 per minute. They were not uniformly rhythmic, and were no longer diphasic.

10th September.—The ventricles were beating wholly irregularly at a rate of about 60 per minute. Electrocardiograms showed small irregular auricular deflexions at a rate of about 457 to 500 per minute. Many of the ventricular beats were represented by atypical deflexions. None of these atypical beats were premature; on the contrary, each was preceded by a long diastolic pause. In one part of the record three

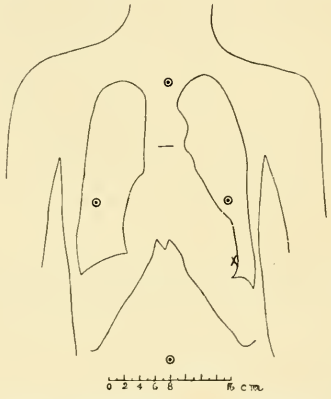


Fig. 50.—Orthodiagram of heart and aorta at end of tranquil inspiration. Case II.

atypical beats occurred in series. The digitalis was stopped—he had taken 1575 minims in the course of 32 days. An orthodiagram (Fig. 50) showed that all the diameters of the heart were considerably enlarged; the long diameter from the cavo-auricular junction to the apex was 18·3 cm.; the curves of the right auricle and left ventricle extended outwards 6 and 9 cm. respectively from the mesial line. The left auricular curve was absent; the pulmonary and aortic curves on the left side were somewhat more prominent than normal.

11th September.—The auricles were still in fibrillation. In electrocardiograms the rate of the auricular deflexions was about 476 per minute; the ventricular rate was about 65 per minute, with intersystolic periods of 0·73, 0·99, 1·29, 1·22, 0·85, 0·67, 0·84, 0·91, 0·64, 0·71, 1·01 second.

14th September.—The rhythm of the ventricles has remained wholly

irregular. Electrocardiograms recorded on the 12th and again to-day showed auricular fibrillation.

15*th September.*—The ventricles were still irregular at a rate of 76 per minute. The maximum systolic pressure was 140, the diastolic pressure 90 mm. Hg.

16*th September.*—Continuous irregularity of the ventricles—the rate was 86 per minute. Administration of mercury and iodide was commenced.

19*th September.*—The pulse was wholly irregular, with a rate of 88 per minute.

About a fortnight later the patient was not so well; the pulse-rate had become more frequent, the administration of mercury and iodide was stopped, and digitalis was again administered. On the 22nd October, the last occasion on which the patient was seen, the auricles were still in fibrillation.

Case III.—Auricular Flutter at a Rate of 320 per Minute; Onset of Fibrillation under the Influence of Strophanthin; Subsequent Return to Physiological Rhythm; Death Ten Days after the Flutter.[1]

A cabman, aged 37, married, but without children, was admitted to the Royal Infirmary on the 25th February 1912, under the care of the late Dr. George A. Gibson. The patient was complaining of swelling of the abdomen, and of an uncomfortable feeling after taking food. He had suffered from acute rheumatism three times—when 15, 22, and 27 years of age—but denied having suffered from venereal disease. He was a moderate drinker. For about six weeks he had been complaining of shortness of breath, especially on exertion, but he had not experienced any pain, palpitation, or faintness. On admission he was listless and markedly cyanotic, but not dropsical. He was 5 feet $6\frac{1}{4}$ inches in height, and weighed 137 lb. The heart was enlarged, the apex-beat being in the sixth left intercostal space, $4\frac{1}{2}$ inches from the mid-sternal line. A loud systolic and a softer diastolic mitral murmur were constantly audible; a rough presystolic mitral murmur could usually be heard, but it was not constant. The arterial pulse was rhythmic and not unduly frequent, the walls of the radial arteries were not palpably thickened, and the maximum systolic pressure did not exceed 110 mm. Hg. Electrocardiograms recorded on the 28th February and on the

[1] "Auricular Flutter," *Edin. Med. Journ.*, Edin., 1912 (N.S.), ix., 485.

7th March 1912 showed that each auricular beat was followed by a ventricular contraction. The auricular and ventricular deflexions were similar to those in Plate XI., Figs. 56, 57, and 58. The patient was treated with strychnine and digitalis, and was discharged on the 28th March 1912 much improved.

He was re-admitted on the 21st July 1912 under the care of Professor Wyllie. The patient was then complaining of shortness of breath and swelling of the feet and ankles. He sometimes had palpitation at night, but had not suffered any pain. On several occasions he had felt faint, and had to grasp a railing to steady himself.

The apex-beat was situated $5\frac{1}{2}$ inches to the left of the mid-sternal line. Considerable pulsation was visible, both in the epigastrium and in the jugular veins on the right side of the neck. The same mitral murmurs were audible as when the patient was in hospital previously. The arterial pulse was rhythmic, and its rate was 94 per minute. Examination of the lungs revealed no abnormality; the urine was scanty and contained albumin.

To Professor Wyllie's resident physician, Dr. J. M. Murray, I am indebted for the following clinical notes of the patient's progress:— He was given a milk diet, and 5 minims of tincture of strophanthus and 10 grains of potassium citrate were administered thrice daily. On the 23rd July he began to take 10 grains of diuretin thrice daily. On the 24th July the patient was feeling better. He passed 148 ounces of urine, and the œdema had lessened. His pulse-rate had varied from 84-96 per minute. On the 26th July there was some sickness, and tincture of digitalis in 5-minim doses thrice daily was substituted for strophanthus. The rate of the pulse was 88, and its rhythm was regular.

On the 31st July the pulse was at a rate of 76, and regular. The patient felt very well. The œdema had disappeared and the digitalis was stopped. By the 9th August he had improved steadily and was feeling very well, but he was still kept in bed, and he continued to take potassium citrate and diuretin. His pulse-rate varied from 68-72 per minute. On 15th August he was allowed to get up for a short time. The pulse-rate had been 68-72 per minute. On the 17th and 18th he was feverish (temperature 100·0° F.). On the 20th August the legs were again œdematous; his pulse-rate had been about 80-98 per minute, and he was again confined to bed. On the 1st September his pulse-rate was 92-98, and he commenced to take 10 minims of tincture of digitalis thrice daily.

On the 7th September the patient's condition was decidedly worse, and hardly any pulse could be felt at the wrist. On the following day his condition was much the same.

On the 9th September his arterial pulse-rate was 160 per minute; the beats were rhythmic and equal, but of small volume. Marked pulsation was seen in the jugular veins, and tracings taken at 1.15 P.M. revealed a ventricular venous pulse (Fig. 51). He vomited once early in the afternoon. Electrocardiograms (Plate XI., Figs. 52, 53, and 54) taken at 3 o'clock showed that the auricles were beating rhythmically at a rate of 320, while the ventricular beats were rhythmic at a rate of 160 per minute. From 5.28 until 5.30 P.M. the ventricular rate, estimated from a venous pulse tracing, was constantly 162·1 per minute. At 5.31 P.M. 0·001 gramme of strophanthin (Boehringer) was given intravenously, and a polygraphic record was taken every minute until

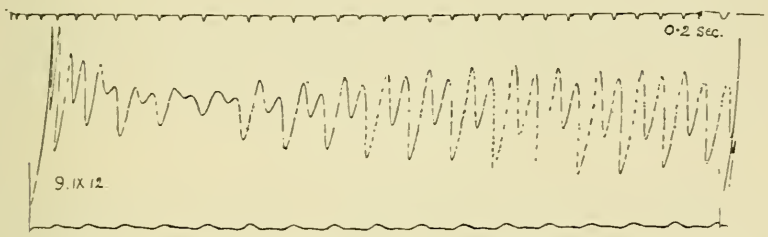


FIG. 51, Case III.—Jugulo-carotid and brachial tracings. The arterial pulse-rate is 159·5 per minute. The jugular pulse is of the ventricular form.

6.1 P.M. This record showed that the ventricular rate fell to 155·2 at 5.33, varied from 153·8 to 162·1 until 5.41, and remained constant at 157·9 from 5.42 until 5.59. At 6 P.M. the rate was 162·1, and at 6.1, half an hour after the injection, the rate was 161·3. At 6.3 P.M. pressure upon the right vagus retarded the ventricles for a few seconds, after which they resumed their former rate. At 8 P.M. the arterial pulse had fallen to 128 per minute, and its rhythm was irregular. He vomited several times during the evening, and complained of some pain in the muscles of the arm into which the injection had been given. He slept poorly.

On the following day (10th September) he felt better, though still troubled with cough and dyspnœa. The lungs presented no signs of œdema. The arterial pulse was less frequent (96-104 per minute) and of larger volume, but it was wholly irregular for the first time. An electrocardiogram (Fig. 55) showed both the disorderly action of the ventricles and the small irregular quick deflexions characteristic of auricular fibrillation. The auricular deflexions were at a rate of about

400 per minute. The dose of tincture of digitalis was increased to 15 minims thrice daily.

On the succeeding day (11th September) he looked and felt decidedly better. He had slept well and the feet had become less œdematous. The arterial pulse was perfectly rhythmic at a rate of about 90 per minute. Electrocardiograms, recorded by derivations I., II., and III., showed that each auricular beat was followed by a ventricular contraction, and that the *As*—*Vs* interval was 0·15 second (Figs. 56, 57, and 58). In these electrocardiograms the auricular and the ventricular deflexions were of the same form as those in the records taken six months previously; but on comparing the auricular deflexions when the auricles were in flutter (Figs. 52, 53, and 54) with those when they were beating slowly (Figs. 56, 57, and 58) it is evident that the former, especially as recorded by derivations I. and III., were of abnormal form. When the auricles were in flutter, the stimulus for each contraction was therefore initiated at an abnormal site.

On the 13th September he remained fairly well, and his pulse was rhythmic at a rate of 98 per minute. On the 14th and 15th he was obviously worse, his face having an icteric tint. His pulse was 90-100, and rhythmic. In electrocardiograms recorded on the 14th, each auricular beat was seen to be followed by a ventricular contraction. On the 16th the right knee became swollen and painful. He was given aspirin thrice daily, and on the 17th the joint had improved. His pulse was rhythmic at a rate of 100-102 per minute. On the 18th the pulse was still rhythmic, and at a rate of 88-96; the dose of digitalis tincture was reduced to 5 minims thrice daily. On the same evening, however, he was suddenly seized with severe breathlessness. Examination of the chest revealed evidence of a pleural effusion. After 25 ounces of fluid had been withdrawn he was somewhat relieved for a time, but the dyspnœa soon recurred. It was not relieved by $\frac{1}{100}$ grain of strophanthin given subcutaneously, and the patient died on the following morning.

The pathological appearances of this heart have been described on p. 30.

Case IV.—Auricular Flutter at a Rate of 256-270 per Minute in an otherwise Healthy Heart; Recovery.[1]

A railway clerk, aged 21, had enlarged glands and sinuses in the neck since he was seven years old, but otherwise had enjoyed good

[1] "Auricular Flutter," *Edin. Med. Journ.*, 1912 (N.S.), ix., 485.

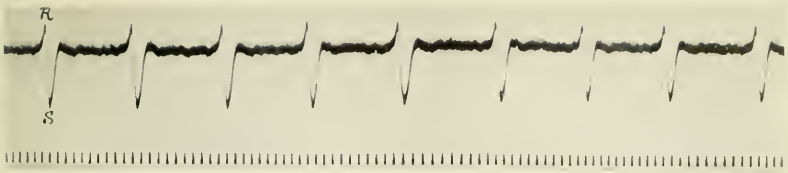


Fig. 52.—Auricular flutter. Derivation I. 9, ix. 1912.

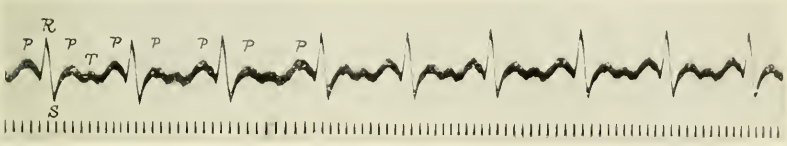


Fig. 53.—Auricular flutter. Derivation II. 9, ix. 1912.

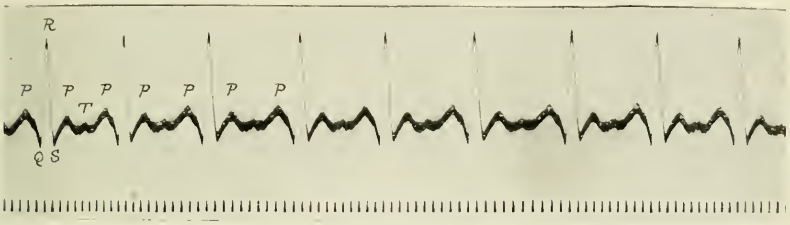


Fig. 54.—Auricular flutter. Derivation III. 9, ix. 1912.

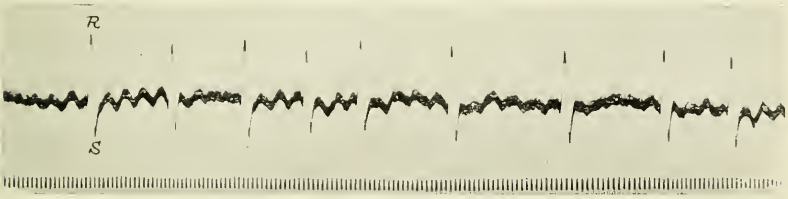


Fig. 55.—Auricular fibrillation. Derivation II. 10, ix. 1912. Nineteen hours after strophanthin intravenously.

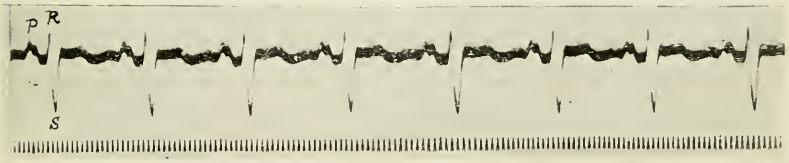


Fig. 56.—Normal rhythm. Derivation I. 12, ix. 1912.

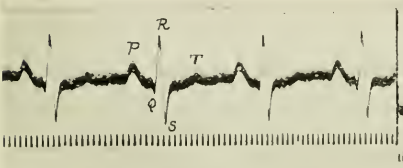


Fig. 57.—Normal rhythm. Derivation II. 11, ix. 1912.

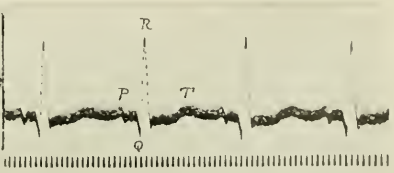


Fig. 58.—Normal rhythm. Derivation III. 11, ix. 1912.

Electrocardiograms from Case III. In Fig. 55 the tension of the string was adjusted so as to give a deflexion of 1·5 cm. for 1 millivolt. In the remaining records 1 cm. = 1 millivolt. The time record is 28·57 per second. (*Edin. Med. Journ.*, N.S., vol. ix.)

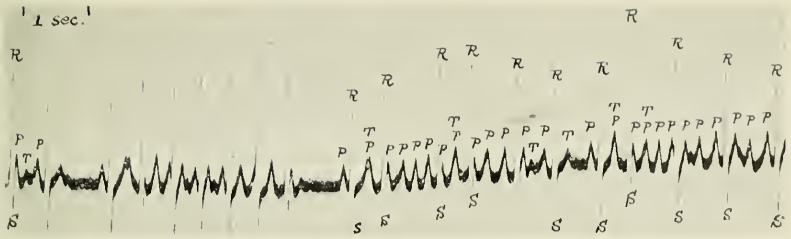
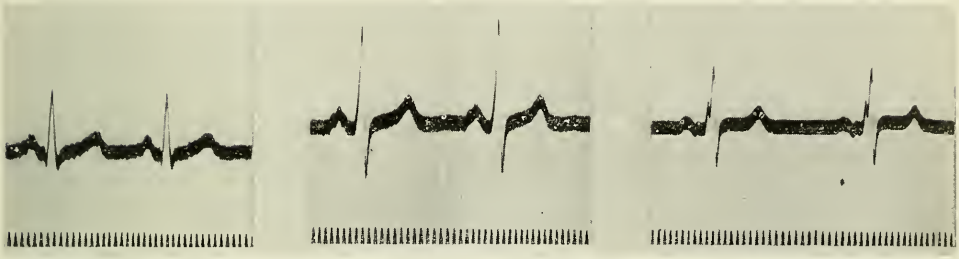


FIG. 59.—Auricular flutter. Case IV. Derivation II. 1 cm. = 1 millivolt.
(*Edin. Med. Journ.*, N.S., vol. ix.)



FIGS. 60, 61, 62.—Electrocardiograms by derivations I., II., and III., after restoration of the normal rhythm in Case IV. 1 cm. = 1 millivolt.

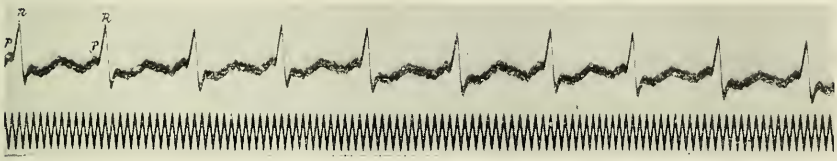


FIG. 63.—Auricular flutter at a rate of 285·6 per minute. The ventricles are responding rhythmically at a rate of 142·8 per minute. The auricular deflexions are alternately merged in *T*, and precede *R* by 0·07 second. Case XI. Derivation II. 1 cm. = 1 millivolt.

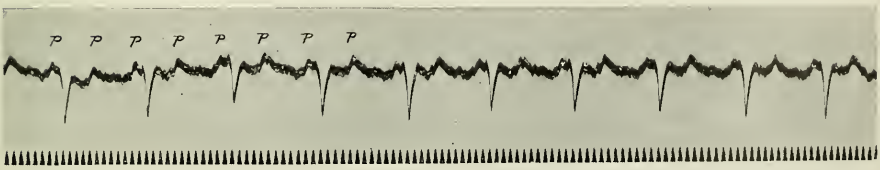


FIG. 64.—Auricular flutter. Case XI. The rate per minute of the auricles is 285·6, while that of the ventricles is 142·8. Derivation III. 1 cm. = 1 millivolt.

health, and had never suffered from acute rheumatism, chorea, scarlatina, or syphilis. In May 1911 he suffered from palpitation for two or three weeks whenever he exerted himself. He could then feel his heart beating forcibly and at a great rate. The attack of palpitation might pass off in a few minutes, or might persist for two or three days, and would then prevent him from sleeping. In November 1911 the palpitation recurred, and was associated with vertigo, which was even more troublesome. Both symptoms used to appear whenever he underwent any unusual exertion; for example, when he ran upstairs, or after a wrestling bout, but at all other times he felt perfectly well. On one occasion, after running, he nearly fainted. He never experienced any pain or dyspnœa. He had been off work for fourteen weeks when he was admitted to the Royal Infirmary under the care of Professor Russell in May 1912.

Electrocardiograms taken on the 10th May 1912 showed that the ventricles were beating irregularly in response to supra-ventricular stimuli. The ventricular periods estimated from an electrocardiogram were 0·45, 0·42, 0·35, 0·77, 0·43, 0·49, 0·45, 0·45, 0·49, 0·39, 0·49, 0·35, 0·45, 0·80, 0·42, 0·49, 0·45, 0·43, 0·45, 0·49, 0·42, 0·35 second. These figures indicate that the ventricles were usually beating at a rate of about 122-133 per minute, and that there was a comparatively long diastolic pause after a group of several frequent beats. The ventricular arrhythmia is illustrated in Fig. 59, where each of the longer pauses is approximately equal to the sum of the two antecedent ventricular periods. The auricular rhythm was also irregular. There were series of 8-10 auricular beats occurring wholly or almost rhythmically at a rate of 256-270 per minute, and terminating in a diastolic pause of about 0·7 second duration (Fig. 59). The irregularity of the ventricles was due in part to the auricular irregularity, and more especially to the occurrence of a comparatively long auricular diastole every now and again. The ventricular irregularity, however, was also due to the fact that even when the auricles were beating fairly rhythmically successive ventricular contractions occurred in response to a varying number (for example, 4, 2, 3, 2, etc.) of auricular beats. The auricular flutter was not arrested by pressure on the right vagus.

The patient stayed in hospital for five weeks, and a fortnight after his return home he was able to resume his work. When he was again examined four months later, he was still feeling perfectly well. His heart was of normal size, without any sign of a valvular lesion, and the rhythm was a physiological one (Plate XII., Figs. 60, 61, and 62).

The flutter in this case differs from that in most other recorded cases in not being continuous, but, on the contrary, intermittent; and further, the auricular deflexions during the flutter were monophasic and of the same form as when the beats were physiological.

Case V.—Transitions between Auricular Flutter, Fibrillation, and a Combined Form of Auricular Activity.[1]

The patient, a miner, aged 38, was a married man with four healthy children. His only antecedent illness was an attack of acute rheumatism, lasting for four months, twelve years previously. At times he had been immoderate in the use of alcohol. For three weeks before his admission to hospital he had experienced shortness of breath, pain at the lower part of the sternal region, and a choking sensation in the same region, "as if a lump were there which he could not swallow." The act of deglutition, however, was not attended by any difficulty or pain, and his appetite was good. He had not suffered from palpitation or faintness, nor had there been any dropsy.

On admission to the Royal Infirmary under the care of Professor Russell, the patient was found to be a rather poorly-nourished man of 5 feet 7 inches, weighing 143 lb. His complexion was pale, and although the ears and nose were of a red tint there was no cyanosis. The cardiac impulse was widespread, forcible, and usually irregular, the point of maximum impulse being in the sixth left intercostal space 4 inches from the mid-sternal line. The only endocardial murmur was a rough diastolic murmur, of mitral origin, which became audible during the longer ventricular pauses. The walls of the radial and brachial arteries presented a moderate degree of diffuse thickening, and the systolic arterial pressure was equal to 140 mm. Hg. The lungs, abdomen, urine, and nervous system presented no abnormal features.

During the first four days the patient was in hospital his ventricular action, although sometimes rhythmic, was usually irregular, and was constantly accelerated. When the ventricles were beating rhythmically the auricles were in flutter, there being a ratio of As : Vs : : 2 : 1. When the ventricles were irregular the auricles were either in fibrillation or in a form of activity indicating flutter combined with fibrillation. During his stay in hospital the patient took fourteen Nativelle's granules, each containing $\frac{1}{240}$ grain of digitaline. In the course of a

[1] "Further Observations on Auricular Flutter," *Quart. Journ. of Med.*, Oxford, 1913-14, vii., 1.

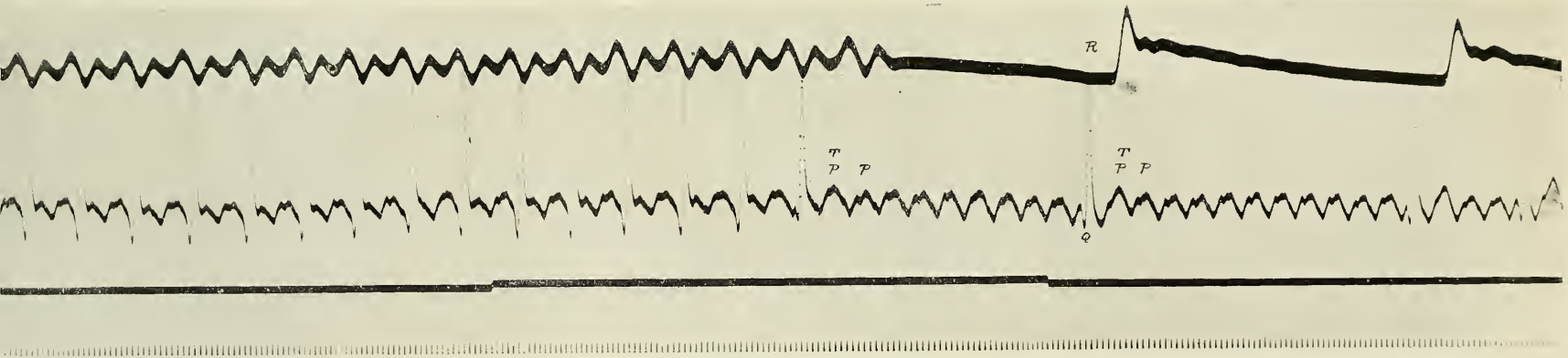


Fig. 65.—Brachial pulsations and electrocardiogram by derivation III. Case V. The signal indicates the time during which pressure was applied to the right vagus. This retards the ventricles, but the auricular flutter persists at a rate of 339—342 per minute. 1·5 cm. = 1 millivolt. (*Quart. Journ. Med.*, vol. vii.)

few days he obtained entire relief from all his symptoms, and on the eighteenth day, when his pulse was still irregular and accelerated, and when he was still taking one granule daily, he felt perfectly well and insisted on returning home. Thus no opportunity was afforded of observing the nature of the auricular action after the digitaline was discontinued.

1. *The Auricles.*—Three forms of auricular action were observed—flutter, fibrillation, and a combined form.

(a) *Flutter* was recorded by derivations II. and III. By both derivations the auricular deflexions were rhythmic, of large amplitude (200 microvolts), of constant form except when distorted by ventricular deflexions, and usually occurred at a rate of 339-343 per minute. On one occasion, immediately after the auricles had passed from fibrillation into flutter, the auricular rate was as high as 377·2 per minute.

When the ratio of As : Vs was 2 : 1 the auricular deflexions were distorted by those of ventricular origin, and thus the true nature of the heart's action was somewhat obscured. The auricular flutter was revealed clearly, however, when the ventricles were retarded by means of pressure on the right vagus in the neck. This is illustrated in Plate XIII. The record starts with fifteen rhythmic ventricular beats at a rate of 169·7 per minute. The corresponding beats of the brachial pulse are hyperdicrotic but not alternating. The auricular rate, meanwhile, is exactly twice that of the ventricles, namely, 339·4 per minute. The summits of successive *P* deflexions fall 0·10 second after the commencement of *R* and synchronously with the summit of *T* respectively. Pressure upon the right vagus slows the ventricles markedly. The longer of the two prolonged ventricular diastoles lasts for 1·75 second. While the ventricles are retarded the auricular deflexions continue rhythmically at a rate of 342·8 per minute.

The initial auricular deflexion is upwards; there are two summits, of which the second is the higher. The curve then descends below, and subsequently regains, the level at which *Q* starts, but the diphasic form of the auricular deflexions is less obvious than in Cases I. and II.

(b) *Fibrillation.*—The auricular deflexions, as a rule, occurred at a rate of about 415-467 per minute. They were either small and markedly irregular in form and rhythm (fine fibrillation) or larger (50-150 microvolts) and less irregular in form and rhythm (coarse fibrillation) (Plate XXI., Fig. 106).

(c) *The simultaneous occurrence of flutter and fibrillation* was characterised by auricular deflexions at a rate of about 367 per

minute, of fairly constant but not diphasic form, of considerable amplitude (about 150 microvolts), and almost wholly rhythmic. Between this form and coarse fibrillation on the one hand and flutter on the other there were no hard-and-fast lines of distinction.

2. *The Ventricles.*—When the auricles were fluttering the ventricles beat rhythmically at a rate of 169·7-171·6 per minute, the auriculo-ventricular ratio being constantly 2 : 1. The arterial pulse was small and hyperdicrotic, as in the first portion of Fig. 65, but it was never alternating. On one occasion, immediately after the cessation of auricular fibrillation, a ventricular rate of 188·6, with an auricular rate of 377·2 per minute, was recorded (Plate XXI.). When the auricles were in fibrillation the ventricular contractions were arrhythmic and less frequent, their rate varying from 76 to 107·1 per minute. When the auricular action was a combination of flutter and fibrillation the ventricular rhythm was, in the main, wholly irregular, but occasionally the length of some of the inter-ventricular periods was an exact multiple of the average *P-P* interval, suggesting that a regular auricular rhythm predominated now and then. Thus in one record, where the average *P-P* interval was about 0·14 second, the inter-ventricular periods were—

0·59, 0·42, 0·69, 0·89, 0·80, 0·70, 0·42, 0·42, 0·45, 1·12, 0·98, 0·45, sec.
$= 0{\cdot}14 \times$ 3 5 3 3 8 7

In another record the rate of the auricular deflexions was about 367·1 per minute, and the inter-ventricular periods were—

0·63, 0·64, 0·73, 0·94, 0·60, 0·63, 0·87, 0·45, 0·91, 0·63 second.
2·0 2·10 1·99

The grouping of the beats is almost exactly constant, which suggests that there is a regular dominant rhythm of the auricles.

Case VI.—Auricular Flutter at a Rate of 333-340 per Minute; Subsequent Onset of Auricular Fibrillation which Persisted until a Fortnight before the Patient's Death.[1]

The patient, aged 59, had no antecedent history of acute rheumatism or valvular disease, but for several years he had been subject to paroxysmal attacks of tachycardia in which the ventricles beat irregularly at a rate of about 158 per minute; and there had been a syncopal seizure on one occasion. From each of these attacks the patient had made a rapid and speedy recovery after the heart had

[1] See also Mackenzie, *Diseases of the Heart*, third edition, Lond., 1913, Appendix, Case 63.

regained its normal rhythm. The last attack of flutter began five months before the patient's death. Dyspnœa, palpitation, Cheyne-Stokes breathing and insomnia, were the dominant symptoms. The auricles were beating rhythmically at a rate of 333-340 per minute. The auricular waves in the jugular veins were of large size (Fig. 66). Meanwhile the ventricular rate was about 110 per minute, and the rhythm was notably irregular, for although the auriculo-ventricular

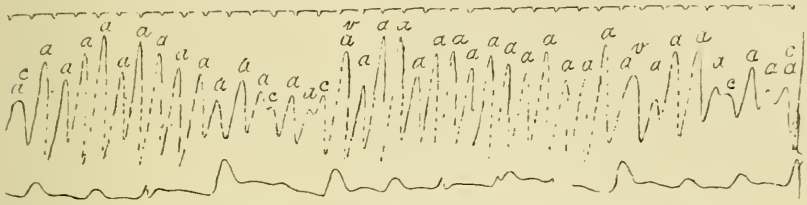


Fig. 66.—Auricular flutter at a rate of 350 per minute. The ventricular responses to every third auricular beat are of unequal strength. Case VI., jugulo-carotid and brachial tracings.

ratio was often 3 : 1, it frequently varied between 2 : 1, 3 : 1, and 4 : 1. The ventricles could not be retarded by compression of the vagus on either side.

The symptoms of cardiac failure becoming more urgent, the daily dose of digitalis tincture was increased to 1 drachm. Four days later, the large auricular waves disappeared from the jugular veins, the venous pulse acquired the ventricular form, and the arterial pulse became

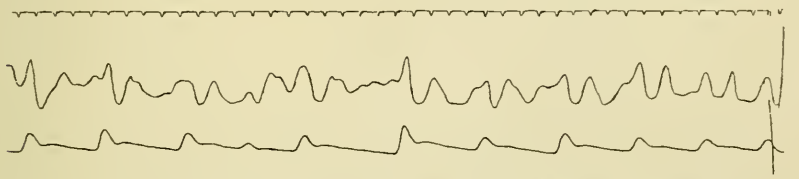


Fig. 67.—Jugulo-carotid and brachial tracings from Case VI., after development of auricular fibrillation.

wholly irregular (Fig. 67). The auricular flutter had apparently passed into fibrillation. Subsequently the ventricles became dilated, the mitral valve incompetent, and the urine scanty and albuminous. The auricles apparently remained in fibrillation for three and a half months. Thereafter, during the fortnight preceding the fatal issue and while the administration of digitalis was still being continued, periods of auricular fibrillation, with ventricular arrhythmia, alternated with others in which the venticles were beating rhythmically at a rate of 64-80 per minute.

Case VII. — Exophthalmic Goitre; Extrasystolic Arrhythmia; Probable Onset of Auricular Flutter; Terminal Auricular Fibrillation; Degenerative and Inflammatory Changes in the Auricular Musculature.

Mrs. H., aged 41 years, suffering from exophthalmic goitre, was under my care in the Royal Infirmary in 1911. The thyroid had been enlarged for fourteen years. Seven weeks before her admission to hospital, exophthalmos, tachycardia, nervousness, and emaciation became pronounced. There was brown pigmentation of the skin, particularly of the abdomen. The heart was dilated and feeble. The ventricular rate was usually about 100 per minute.

For some days after her admission to hospital, records showed occasional extrasystoles which were certainly supra-ventricular, and

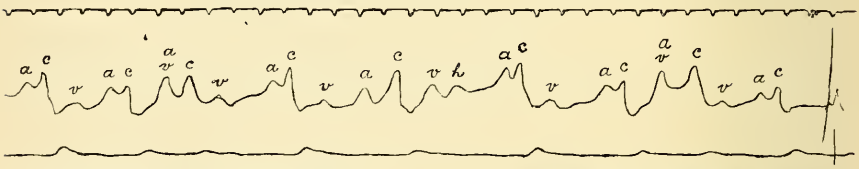


Fig. 68.—Sinus irregularity and two extrasystoles (third and eighth beats). Jugulo-carotid and brachial tracings from Case VII.

probably auricular, in origin. The inter-systolic periods in a tracing from the apex-beat were—

0·60, 0·60, 0·40, 0·75, 0·64, 0·40, 0·70, 0·65, 0·60, 0·60 second.

1·20 1·15 1·10 1·20

These figures indicate that the post-extrasystolic pause was not fully compensatory and that the second beat after each extrasystole had a longer diastolic pause (0·64 and 0·65 second) than that of the other physiological beats (0·60 second). The *a-c* interval of the physiological beats was 0·13 second. A week later extrasystoles became more numerous, and often occurred in series of from two to six or eight. Some of these extrasystoles were of ventricular origin, others were supra-ventricular (Fig. 68), and in some instances a ventricular extrasystole was followed immediately by an auricular one. At the same time there was also marked sinus irregularity, and this together with the extrasystoles rendered the arterial pulse markedly irregular. In one record the intersystolic periods, estimated from a brachial tracing, were 0·86, 0·86, 0·52, 0·40, 0·40, 0·40, 0·45, 0·46, 0·50, 0·70, 0·70, 0·50, 0·89, 0·90, 0·50, 0·40, 0·64, 0·44, 0·44, 0·50, 0·42, 0·92, 0·76, . . . 0·45,

0·40, 1·30, 0·85, 0·74, 0·74, 0·50 second. The length of the *a-c* interval was constantly varying from 0·15, 0·17, or 0·18 to 0·25 second.

A fortnight later the extrasystolic arrhythmia became replaced by an almost absolutely rhythmic action of the ventricles at a rate of 113-120 per minute, this being associated with a slight degree of pulsus alternans (Fig. 69). The auricles were probably fluttering at a rate of 226-240 per minute. This condition lasted for two days. On the third day, when the patient was obviously dying, the radial

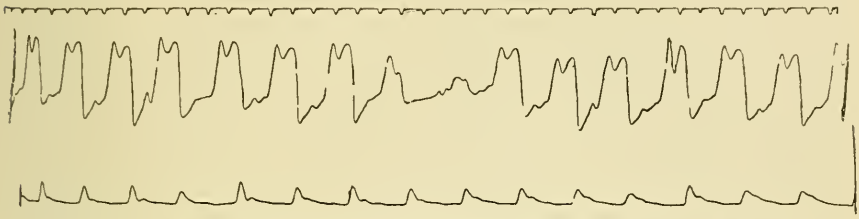


FIG. 69.—The ventricular rate is 114 per minute, and there is slight alternation of the pulse. Apical and brachial tracings. Case VII.

pulse could hardly be felt, the rate of the ventricles rose to 171-224 per minute and their rhythm became extremely irregular (Fig. 70). An intra-venous injection of digitalin failed to retard the ventricles, and the patient died the same evening. Although no electrocardiograms or satisfactory jugular tracings could be obtained during the last three days of life, the sequence of extrasystolic arrhythmia, rhythmic ventricular tachycardia, and gross ventricular irregularity, suggests that

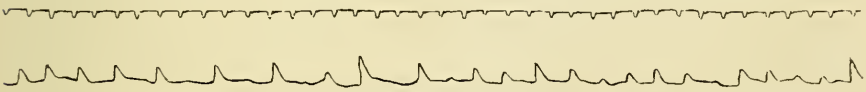


FIG. 70.—Pulsations of the abdominal aorta at a rate of 187 per minute. Case VII., twelve hours before the patient's death.

the extrasystolic arrhythmia passed into auricular flutter, and this in turn into fibrillation.

The conditions found at the post-mortem examination have been described on p. 32.

CASE VIII.—POST-INFLUENZAL TACHYCARDIA PERSISTING FOR SOME MONTHS; IMPROVEMENT UNDER DIGITALIN; PATIENT WELL FOUR YEARS LATER.

The patient was a lawyer, of middle age, whose professional work had been arduous, and who had been in the habit of taking little leisure or recreation. His previous health had been uniformly excellent, and

he never had occasion to consult a doctor until he became laid up with influenza early in January 1905.

When I saw him for the first time on the last day of March 1905 he stated that ever since the influenzal attack he had been greatly troubled with nervousness, insomnia, palpitation, and perceptible irregularity of the heart. He did not look seriously ill, and was neither cyanotic nor dropsical. His heart was not enlarged and no valvular murmurs were audible, but the first sound at the mitral area was reduplicated. The lungs and abdominal viscera were healthy. The patient was taking strophanthus and strychnine, yet his ventricular rate was usually about 125-140 per minute; the rhythm was sometimes, but not constantly, regular. At times the ventricular rate rose to about 170 per minute, and the pulse was then alternating.

The patient was extremely nervous about himself, slept poorly in spite of hypnotics, and was liable to attacks of sweating and of retching. His condition continued unchanged for eighteen days, when $\frac{1}{100}$ grain of digitalin four times daily was substituted for the strophanthus and strychnine. The pulse-rate was then 134 per minute, and the radial tracing showed frequent rhythmic alternating beats in groups which were separated from one another by a pulse period equal to two of the frequent beats. The ventricles were probably responding successively to . . . 2, 2, 4, 2, 2, 2, 4, 2, 2, 2, 2, . . . auricular beats.

After the patient had taken digitalin for three weeks his condition was greatly improved. His appetite was good, he slept soundly, and he was able to be out for a drive each day. His pulse was then rhythmic at a rate of 100 per minute. Four years later the patient was reported to be in excellent health and able for all his professional work.

Case IX.—Probable Onset of Auricular Flutter at a Rate of 454 per Minute during Chloroform Anæsthesia.[1]

Male, aged 19, under the care of Mr. Cathcart in the Royal Infirmary. Records were taken before and during an operation for osteomyelitis of the femur. Twenty-five minutes before the anæsthetic was given the auricles and ventricles were beating rhythmically at a rate of 81 per minute. Seventeen minutes after the commencement of administration of chloroform they were beating rhythmically at a rate of 75 per minute. Thirteen minutes later (1.33 P.M.) the pulse was

[1] "Auricular Flutter," *Edin. Med. Journ.*, Edin., 1912 (N. S.), ix., 485.

markedly hyperdicrotic and alternating, and its rate was 166. At 1.34 and 1.35 the rates were 194 and 176 respectively; the rhythm was still regular, and alternation was pronounced. At 1.36 the rate of the pulse had risen to 227-230·7 per minute; it was still rhythmic and alternating. The jugulo-carotid tracing presented a continuous series of large waves at a rate of 454 per minute. The auricles were probably in flutter. The rhythmic tachycardia persisted at about the same rate for three minutes. The patient then became cyanosed, the administration of the anæsthetic was stopped, and artificial respiration was performed. At 1.40 the pulse, which had been alternating and rhythmic, became wholly irregular. The pulse periods, estimated from the tracing, were 0·32, 0·30, 0·52, 0·56, 0·35, 0·40, 0·38, 0·30, 0·40, 0·44 second. At that time the auricular flutter had apparently passed into fibrillation. Two minutes later, however, the auricles and ventricles

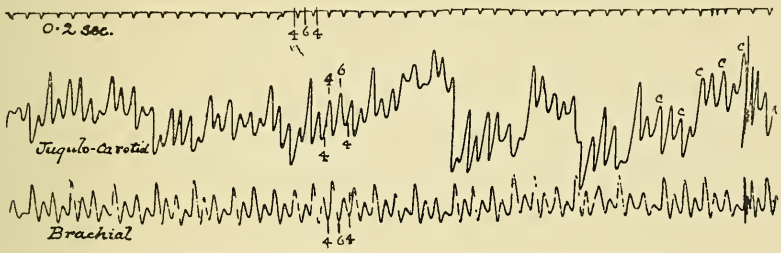


Fig. 71.—Rhythmic tachycardia at a rate of 225 per minute. Case IX.

had resumed their rhythmic action at a rate of 125 per minute, and this condition lasted until the record ceased. The patient's subsequent progress was uneventful.

In Case II., with undoubted auricular flutter, the arterial pulse was frequent, rhythmic, and alternating, and was associated with a jugular pulse of ventricular form. The same phenomena were recorded in the following case, which is therefore regarded as auricular flutter.

Case X.—Arterio-sclerosis with Heart Failure; the Auricles were probably Fluttering at a Rate of 262 per Minute.[1]

The patient, a grocer, aged 60 years, was seen on the 5th September 1912. He was a married man, of careful habits, and he had not been intemperate. He had been a very active man, leading a busy life, until at the age of 59 he began to suffer from palpitation. He had never experienced any cardiac pain. Dropsy had become progressively worse

[1] "Auricular Flutter," *Edin. Med. Journ.*, Edin., 1912 (N. S.), ix., 485.

in spite of rest in bed, digitalis, and diuretin. When I saw the patient, one year after his illness began, he was confined to bed. His heart was moderately enlarged, the right border being in the right lateral sternal line, and the apex-beat being in the fifth left intercostal space half an inch outside the mammillary line. On auscultation a high-pitched systolic mitral murmur was audible. The second sound was loudly accentuated. A considerable degree of pulsation, which was of ventricular form, was visible in the jugular veins on the right side of the neck (Fig. 72). The walls of the arteries were thick, and the systolic pressure was 275 mm. Hg. The ventricles were beating rhythmically at a rate of 131 per minute, and the arterial pulse was alternating (Fig. 72). The auricles were probably fluttering at a rate of 262 per minute. The patient died about one month after the record in Fig. 72 was obtained.

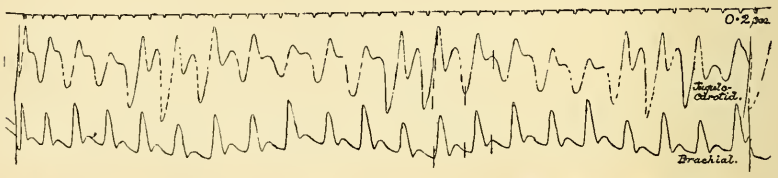


Fig. 72.—Rhythmic tachycardia at a rate of 131 per minute, with a ventricular venous pulse. Case X.

Case XI.—Auricular Flutter at a Rate of 285 per Minute; Heart Failure; Death.

The patient, aged 65, who was under the care of Dr. Lovell Gulland in the Royal Infirmary, had a persistent and rhythmic tachycardia at a rate of about 140 per minute for twelve weeks before his death. No murmurs were audible. Digitalis, even although given in large doses, failed to retard the heart. Œdema and bilateral hydrothorax developed, and the patient gradually sank, and died eighteen days after the records in Plate XII., Figs. 63 and 64, had been obtained. The post-mortem appearances have been described on p. 33.

Electrocardiograms by derivations II. and III. are shown in Figs. 63 and 64. The ventricles are beating rhythmically, in response to supra-ventricular stimulation, at a rate of 142·8 per minute. The auricles are beating rhythmically, and twice as fast as the ventricles. One auricular deflexion precedes the initial ventricular deflexion by 0·06-0·07 second; in Fig. 63 the second auricular deflexion is mingled with, and therefore masked by, the terminal ventricular deflexion, but in Fig. 64, by derivation III., both auricular deflexions are visible.

Turning from the cases of my own series, it is found that forty-two other cases of auricular flutter, making a total of fifty-three, have been recorded. A brief summary of the important features of these cases may be given.

Case XII.—Chronic Pericarditis, Heart Failure, and Partial Heart-block; Onset of Auricular Flutter Seven Weeks before Death.

This case I had frequent opportunities of observing while I was attached as clinical tutor to the clinic of the late Dr. George A. Gibson. He recorded the case in 1906.[1] The patient, who was a dairyman aged 44 and had been a heavy drinker, had been breathless for two years before he was admitted to hospital in October 1904. He made a speedy and satisfactory recovery. When re-admitted on the 24th January 1906, he was languid, somnolent, breathless and jaundiced, and had been suffering from palpitation. The right and left borders of the heart respectively were 2½ and 4 inches from the mid-sternal line. When the auricles were in flutter the ventricular rate was usually about 40 per minute, the rhythm was somewhat irregular, and the arterial pulse was weak. The systolic pressure was 93, and the diastolic 72 mm. Hg.

Records taken on the 9th February (Fig. 2 of Dr. Gibson's paper) showed that the auricles were beating rhythmically at a rate of 200 per minute, and that the ventricles responded to every fourth, or every sixth, auricular beat. A portion of another record taken on the same day is shown in Fig. 77. Two days later transient paroxysmal attacks of flutter, with an auricular rate of nearly 350 per minute, alternated with a rhythm that was physiological in every respect except that the rate was somewhat frequent (90 per minute) and an occasional stimulus to the ventricles was b ocked. A few days later the auricular flutter, at a rate of about 350 per minute, became apparently continuous, and nine weeks after the patient's admission to hospital he died somewhat suddenly. The condition of the heart has been described on p. 30.

Case XIII.—The next case in chronological sequence is Morison's[2] case of "jugular embryocardia," observed in 1903 and recorded in 1909.

[1] Gibson, G. A., "A Discussion on some Aspects of Heart-block," *Brit. Med. Journ.*, Lond., 1906, ii., 1113.

[2] Morison, A., "Cardiac Motion as Revealed by the Vivisection of Disease," *Lancet*, Lond., 1909, i., 77; *ibid.*, 1909, i., 39.

In this patient, with mitral disease, the "auricular contractions usually bore to ventricular systole the ratio of 208-228 : 108-114." On auscultation over the jugular area sounds were persistently heard, which in character and rate very strongly resembled those of the fœtal heart. The sounds could not be obliterated by pressure of the stethoscope, and were assumed to be due to right auricular and venous pulsation. During the patient's residence in hospital for four months the condition continued uninterruptedly.

Case XIV.—In the case described by Hertz and Goodhart[1] in 1909, a woman aged 39 years, who was affected with mitral disease and hemiplegia, the auricular rate was almost constantly 234 and that of the ventricles about 80 per minute. The ventricular beats were usually coupled. Atropin did not influence the auricular beats, whereas the ventricular rate rose from 78-84 to 150-170 per minute, two and a half hours after an injection, and fell to 75 seventeen hours later. The rate of the auricles was likewise uninfluenced by physical exercise or by change of posture.

Case XV.—The first of three cases recorded by Rihl.[2] A joiner, aged 55, had been suffering for three months from dyspnœa on exertion. Polygraph tracings and electrocardiograms demonstrated an auricular rate of 285-300, rising on one occasion to 315 per minute. The ventricular rate was 70-150 per minute. The "auricular tachysystole" was not retarded by digalen or by vagus compression, but both induced ventricular slowing. This, when due to digalen, could be abolished transiently by injection of atropin.

Case XVI.—Rihl's second case was a waitress, aged 32, affected with mitral disease. She had been suffering from palpitation and dyspnœa for one year, and from dropsy for one month. An auricular rate of 206-222 persisted probably for about eighteen months, and the patient became markedly dropsical in spite of diuretin, digalen, and other remedies being administered. The ventricular rate was usually 52-72 per minute, but there were occasional paroxysms in which the rate rose to 180-200 per minute. The ventricular rhythm was not uniformly regular. Vagus compression retarded the ventricles only.

[1] Hertz, A. F., and Goodhart, G. W., "The Speed-limit of the Human Heart," *Quart. Journ. of Med.*, Oxford, 1908-9, ii., 213.

[2] Rihl, J., "Hochgradige Vorhoftachysystolien mit Ueberleitungsstörungen und electiver Vaguswirkung," *Zeitschr. f. exper. Pathol u. Therap.*, Berlin, 1911, ix., 277.

Case XVII.—The third case reported by Rihl was a labourer, aged 72, who was seized somewhat suddenly with palpitation, cough, and dropsy. About one year later he was still dropsical; his heart was much enlarged. His auricular rate was 200-214 per minute, but the ventricles were usually beating rhythmically at a rate of 100 per minute. Vagus compression retarded the ventricles. On one occasion there was a paroxysm in which the ventricular rate rose to 207 per minute. When this attack ended the pulse-rate fell suddenly to one-half of its former rate. Infusion of digitalis, caffein, and theobromin were administered; the pulse became slower, and eventually wholly irregular.

Case XVIII.—The first case recorded by Mackenzie [1] was an army officer, born in 1863, who had no rheumatic or specific history. In 1902, during the South African war, he first became conscious of occasional attacks of fluttering in the chest. They did not cause much distress until 1905, when they became very frequent and continued for some months. In 1910 the attacks became numerous and severe, and the patient became very weak. During the attacks, which were provoked by slight exertion, the auricular rate probably varied only from 280-320; the ventricular rate was 130-150, but on one occasion it rose to 290-300 per minute. The face was slightly livid; the sounds of the heart resembled the tic-tac of the fœtal heart. Digitalis produced auricular fibrillation, but later the normal rhythm was restored. The patient continued to take half a drachm of digitalis tincture each day, and gradually regained strength. Eventually the attacks recurred only at rare intervals, and did not last for more than a few minutes. In 1913 he was forced to lead a very quiet life, as exertion induced the rapid heart rate.

Case XIX., recorded by Mackenzie,[2] and subsequently by Turnbull [3] and Lewis,[4] was a man aged 74 who had enjoyed good health until, on waking one morning, he felt ill and was found to have a very rapid

[1] Mackenzie, J., "Digitalis," *Heart*, Lond., 1910-11, ii. 273, Case 37; *Diseases of the Heart*, third edition, Lond., 1913, Appendix, Case 55.

[2] Mackenzie, J., "Digitalis," *Heart*, Lond., 1910-11, ii., 273, Case 38; *Diseases of the Heart*, third edition, Lond., 1913, Appendix, Case 56.

[3] Turnbull, H. Hume, "Paroxysmal Tachycardia accompanied by the Ventricular Form of Venous Pulse," *Heart*, Lond., 1911-12, iii., 89.

[4] Lewis, T., "Observations upon a Curious and not uncommon Form of Extreme Acceleration of the Auricle. 'Auricular Flutter,'" *ibid.*, 1912-13, iv., 171, Case 15.

pulse. About three months later he was cyanosed and dyspnœic. The heart sounds were weak but clear. The ventricles were beating rhythmically at a rate of 150 per minute. The jugular pulsation was of the ventricular form, "though after an occasional pause there was an auricular wave preceding the time of the carotid." Lewis subsequently re-analysed the electrocardiograms and concluded that the auricular and ventricular rates respectively were 280-300 and 140-150 per minute.

A first course of digitalis in considerable doses failed to arrest the paroxysms materially. About two months later the patient took 33 Nativelle's granules in the course of eleven days. On the twelfth day the pulse had fallen to 76, being at times regular, at others irregular. Five days after the digitalis was discontinued the pulse became perfectly regular, and the jugular tracing presented a well-marked auricular wave at its normal instant. It was not until three days later, however, that the dyspnœa and delirium vanished, and the patient really felt well. The heart's rate was then 72 per minute, and except for an occasional extrasystole the rhythm was normal. Thereafter he steadily improved and became able to walk a distance of three miles, mostly uphill, without any distress. Three years later he was still in good health and had no trouble with his heart.

Case XX., recorded by Lewis and Schleiter,[1] and subsequently by Lewis.[2] The patient was a cabinetmaker, aged 28, who had suffered from paroxysmal attacks of auricular fibrillation or flutter. They were accompanied by palpitation, dyspnœa, fainting, fatigue, salivation, sweating, sickness, and great prostration. One attack of flutter lasted for twelve hours. On another occasion an attack of auricular fibrillation lasting for three and a half days became transformed into flutter, and the latter terminated spontaneously. Records showed an auricular rate of 280 with a ventricular rate of 140 per minute.

Case XXI. (Case 2 of the series recorded by Lea[3]).—The patient was a postman, aged 57 years, whose previous health had been excellent. For seven months he had been suffering from dyspnœa, and subsequently he suffered from transient dropsy. He had also complained of precordial pain and "throbbing" over the heart. No murmurs were

[1] Lewis, T., and Schleiter, H. G., "The Relation of Regular Tachycardias of Auricular Origin to Auricular Fibrillation," *Heart*, Lond., 1911-12, iii., 173.

[2] Lewis, T., *ibid.*, 1912-13, iv., 171, Case 16.

[3] Lea, C. E., "Four Cases of Auricular Tachycardia," *Proc. Royal Soc. Med.*, Lond., 1913, vi., Med. Sect., 14.

audible. The auricles and ventricles were beating rhythmically at rates of 260 and 130 per minute respectively.

Under the influence of digitalis the auricular frequency was not lessened, but the ventricular rate fell to one-third of the auricular, and occasionally the ventricular rhythm became irregular owing to the ratio of As : Vs, varying between 1 : 1, 2 : 1, 3 : 1, 4 : 1, 5 : 1, and 6 : 1. Twelve days later the auricles and ventricles were beating with a physiological rhythm at a rate of 65 per minute, but when the patient moved his arms up and down for a little time the auricles relapsed into flutter. After the patient had been under observation for nearly five months the auricles were still fluttering and the ventricles beating at a rate of 130 per minute.

The patient was then leading a quiet life, taking no drugs and keeping in fairly good health. Dr. Lea in a personal communication tells me that the patient died suddenly in June 1913, about nineteen months after the commencement of his illness.

In Case 4 of Lea's [1] series, a woman aged 50, suffering from lymphocythæmia, there were frequent paroxysms during the last five weeks of life, in which the rate of the rhythmically beating ventricles changed abruptly from 90 to 180 per minute. The auricles were probably fluttering at a rate of 360 per minute, and the onset of a paroxysm probably denoted a change of the auriculo-ventricular ratio from 4 : 1 to 2 : 1. At the post-mortem examination the heart was apparently fatty. In a personal communication Dr. Lea informs me he has seen four other cases in which the auricles were probably fluttering.

Case XXII.—Lewis [2] has recorded eight cases of auricular flutter. His first case, a woman aged 50 years, had suffered from acute rheumatism at the age of 23, from some dyspnœa for eleven years, and from notable dyspnœa and palpitation for four months. She also complained of precordial pain. She was cyanosed, but not dropsical; a systolic murmur was audible over the apex of the heart. The attack of rapid heart action had a probable duration of three months. Auricular rates of 300, 314, and 324 were recorded in electrocardiograms. The ventricular rate was usually one-half of the auricular.

After the patient had taken 18 drachms of digitalis tincture in eighteen days, the ventricular rate fell to one-fourth of the auricular.

[1] Lea, C. E., "Four Cases of Auricular Tachycardia," *Proc. Royal Soc. Med.*, Lond., 1913, vi., Med. Sect., 14.

[2] Lewis, T., "Observations upon a Curious and not uncommon Form of Extreme Acceleration of the Auricle. 'Auricular Flutter,'" *Heart*, Lond., 1912-13, iv., 171.

Subsequently auricular fibrillation ensued, and the ventricular rate fell to 79 per minute. The drug was then discontinued. A week later the heart regained its physiological rhythm, and the patient's symptoms were considerably relieved.

Case XXIII.—The second case recorded by Lewis[1] was a clergyman, aged 65, who had been liable to paroxysmal attacks of tachycardia for thirty years. In one attack, persisting for five months, the auricular rate was 266 to 289 per minute. The ventricular rate was usually one-half, but sometimes one-third of the auricular rate. After 485 minims of digitalis tincture had been administered in the course of nine days, the auricles were alternately fluttering and fibrillating; but two days after the drug was discontinued the auricular fibrillation was probably continuous. On the third day the physiological rhythm was regained, and the patient's general condition improved considerably. A few weeks later, however, the auricular flutter returned, and the patient died.

Case XXIV., the third case of Lewis's[1] series, was a clergyman, aged 53, who had experienced attacks of palpitation and rapid heart action for thirty-eight years. The attacks had been more frequent and severe since he suffered from diphtheria at the age of 41. An auricular rate which varied between 264 and 324 was persistent during the seven weeks while the patient was under continuous observation. Meanwhile the ventricles responded to every alternate auricular beat. Neither digitalis nor strophanthus arrested the flutter. Under the former drug, however, the ventricular rate was retarded, and its rhythm became irregular. Compression of the left vagus retarded the ventricles only.

Case XXV.—The fourth case recorded by Lewis[1] was a traveller, aged 62, who developed continuous tachycardia during an attack of "influenza." About two months later, the rate of the auricles and ventricles respectively was 270 and 135 per minute. The patient became cyanosed and breathless, and his heart was enlarged. Digitalis and strophanthus slowed the ventricles, but had no influence on the fluttering auricles. Auricular fibrillation did not ensue. Both right and left vagus compression slowed the ventricles, without affecting the auricles. Six months after the flutter was first recorded the auricles were still fluttering.

[1] Lewis, T., "Observations upon a Curious and not uncommon Form of Extreme Acceleration of the Auricle. 'Auricular Flutter,'" *Heart*, Lond., 1912-13, iv., 171.

Case XXVI.—The fifth case in Lewis's[1] series was a french polisher, aged 60 years, who had been suffering for three months from periodic attacks of breathlessness. He was cyanosed, and the heart and liver were enlarged. The auricular and ventricular rates were 300 and 150 per minute respectively. Compression of either vagus retarded the ventricles only. Digitalis changed the ratio of As : Vs from 2 : 1 into 4 : 1, and thereafter induced auricular fibrillation. The drug was discontinued; the fibrillation apparently persisted for twenty-three days, and thereafter the physiological rhythm was restored. The patient's condition had now become greatly improved; his colour was good, his breathing easy, and the hepatic enlargement had disappeared.

Seven weeks later the auricular flutter recurred, and was observed on several occasions during a period of over four months. The flutter was now uninfluenced by digitalis, but the cardiac pain, dyspnœa, palpitation and dropsy, which had recurred, were relieved. Sixteen days after digitalis was discontinued, and nine days after the patient's discharge from hospital his auricles were fibrillating. One month later the physiological rhythm of the heart was found to have been regained.

Case XXVII.—The sixth case recorded by Lewis[1] was an actor, aged 47, who had suffered for many years from sudden attacks of palpitation under the influence of exercise or emotion, and who was affected with chronic articular gout. The heart was of normal size, but the aortic arch was somewhat dilated. The auricular rate was 330, the ventricular rate 165 per minute.

Case XXVIII., recorded by Mackenzie,[2] and apparently the seventh case of the series reported by Lewis.[1] A healthy looking man, aged 53, had been conscious of intermittent action of his heart since the age of 19, but of late years the "heart attacks" had altered somewhat in character. They might come on unexpectedly, without any assignable cause, and might last for an hour or for several days. The ventricular rate used to rise suddenly from 67 to 130-140 per minute. Electrocardiograms showed an auricular rate of 228, and a ventricular rate of 114 per minute. The area of cardiac dulness was not enlarged, and the

[1] Lewis, T., "Observations upon a Curious and not uncommon Form of Extreme Acceleration of the Auricle. 'Auricular Flutter,'" *Heart*, Lond., 1912-13, iv., 171.

[2] Mackenzie, J., *Diseases of the Heart*, third edition, Lond., 1913, Appendix, Case 59.

sounds were normal. Rest in bed and the administration of digitalin relieved the patient of his symptoms; and about one year later he was reported to have enjoyed good health, though there had been occasional attacks of a few hours' duration.

Case XXIX., the last case of Lewis's[1] series, was a clergyman, aged 52, suffering from dyspnœa and weakness. The heart was enlarged, but no murmurs were audible. The auricles were beating rhythmically at a rate of 260 per minute. The ratio of As : Vs was as a rule 2 : 1, but occasionally 3 : 1 or 4 : 1.

Case XXX.—The case recorded by Hume[2] was a labourer, aged 63, without any rheumatic or venereal history, who for three or four months had been troubled with dyspnœa and giddiness. His heart was enlarged, and there was auscultatory evidence of mitral incompetence. The systolic blood-pressure was 260 mm. Hg. The ventricular rate was about 87, with a ratio of As : Vs :: 3 : 1. A week later varying ratios of 2 : 1, 3 : 1, and 4 : 1 alternated with a more persistent 3 : 1 ratio. Under digitalis the auricular rate remained at 260 per minute, but a continuous auriculo-ventricular ratio of 4 : 1 became established. The ventricular retardation, however, could be relieved by atropin. Auricular fibrillation eventually supervened and became persistent.

Case XXXI.—Goteling Vinnis[3] records two tracings (Figs. 9 and 10 of his paper) showing large auricular waves, not wholly rhythmic, at a rate of 350 per minute, from a case of paroxysmal arrhythmia. The illustrations leave little doubt that the case was one of auricular flutter in which the ventricles responded irregularly.

Case XXXII.—In the first of the two cases recorded by Hay[4] the records extended over a period of about seven years. The patient, aged 46, had been intemperate and had suffered from smallpox and malaria. He was dyspnœic, cyanosed, and dropsical. Auricular flutter at a rate of 240 per minute, with an auriculo-ventricular ratio of 3 : 1 and occa-

[1] Lewis, T., "Observations upon a Curious and not uncommon Form of Extreme Acceleration of the Auricle. 'Auricular Flutter,'" *Heart*, Lond., 1912-13, iv., 171.

[2] Hume, W. E., "A Case in which a High Speed of the Auricles did not produce Tachycardia," *Quart Journ. of Med.*, Oxford, 1912-13, vi., 235.

[3] Vinnis, E. W. Goteling, "De volkomen onregelmatige Hartswerking," *Nederl. Tijdschr. v. Geneesk.*, Amsterdam, 1913, lvii., 501.

[4] Hay, J., "Two Cases of Auricular Flutter," *Lancet*, Lond., 1913, ii., 986.

sional irregularity of the ventricles, was recorded. Alternation of the pulse was pronounced at first, but subsequently disappeared. At a later stage a 4:1 ratio was observed, and still later, under the influence of squill, auricular fibrillation supervened. The patient left hospital greatly improved and began regular work. Four and a half years later the normal rhythm of the heart was found to be restored, and the patient was able to earn a living.

Case XXXIII.—The second case recorded by Hay[1] was a man aged 62, whose heart was much enlarged but who was not dropsical. Auricular rates of 261 and 300 per minute were recorded, with ventricular rates of 50-87 per minute. The auriculo-ventricular ratio was usually 3:1, but this was occasionally interrupted by ratios of 6:1, 4:1. Digitalis did not retard the auricles or induce fibrillation, but it lowered the rate of the ventricles to 31 per minute. The patient continued to take 10 minims of the tincture each night, this dose being sufficient to steady his ventricles. About five months later the rhythm was found to be perfectly normal except for numerous auricular extrasystoles.

A series of over thirty cases has been examined by Mackenzie,[2] who has described fully the clinical features of auricular flutter. In the appendix to his monograph Mackenzie gives a detailed account of fifteen cases (Nos. 55-69) in which the diagnosis was confirmed by the electrocardiograph. Facts regarding some of these cases have been recorded elsewhere either by Mackenzie himself or by other writers (see Cases VI., XVIII., XIX., XXIV., XXV., XXVIII., XXIX., and XXXIII.), but the series includes at least seven new cases.

Case XXXIV.[3]—Male, aged 61, had an attack of influenza and pneumonia, and the heart was found to be rapid and irregular. Five months later he complained of a sense of constriction in the precordial region. His pulse was 144 and its rhythm disorderly; the legs and the bases of both lungs became œdematous. Under the influence of digitalis the patient's condition improved, though he had occasional attacks of tachycardia, and in one attack he felt very faint and almost lost consciousness. On one occasion his auricles were sometimes in flutter, sometimes in fibrillation, and then for a short time the rhythm would

[1] Hay, J., "Two Cases of Auricular Flutter," *Lancet*, Lond., 1913, ii., 986.
[2] Mackenzie, J., *Diseases of the Heart*, third edition, Lond., 1913, 237-250, 428-442.
[3] Mackenzie, J., *ibid.*, Appendix, Case 61.

be perfectly normal. When the auricles were in flutter their rate was 308, while the ventricular rate was 154 per minute.

Case XXXV. (Case 64 of Mackenzie's series).[1]—In this case auricular flutter was the result of an acute infection. The patient, a man aged 47, had been in good health until one evening he became feverish and coughed up some blood-stained sputum. On the next day he was rather breathless and dusky. Three days later there was an attack of great breathlessness, and the pulse became rapid, irregular, and at times scarcely perceptible. Above each clavicle the jugular bulb was seen and felt beating forcibly. Crepitations were audible at the bases of the lungs. About six weeks later he gradually became worse; Cheyne-Stokes respiration developed; the urine became scanty; the legs and abdomen became dropsical; and the patient gradually sank, and died four months after the onset of the illness.

In this case the recorded auricular rate was 240 per minute.

Case XXXVI. (Case 65 of Mackenzie's series).[2]—Female, aged 39, affected with syphilitic aortitis and myocarditis. The patient was cyanosed, breathless, and dropsical. Systolic and diastolic aortic murmurs were audible. After the onset of auricular flutter the general symptoms became worse; no change was found in the area of cardiac dulness or the characters of the murmurs, but the pulse became irregular. The patient died about six months after the initial symptoms of heart failure had appeared and nineteen days after the onset of flutter.

Case XXXVII., recorded by Mackenzie.[3]—Male, aged 63, who had probably suffered from syphilis, and who had been suffering for a few months from cough and attacks of great breathlessness. Electrocardiograms revealed damage to the right branch of the atrio-ventricular bundle. After he had been ailing for about three and a half years he was taken seriously ill with great weakness and breathlessness. He lay, propped up in bed, in a slight stupor and with marked Cheyne-Stokes respiration. The pulse was very rapid, and varied in rhythm. The characters of the jugular pulsations indicated auricular flutter. He gradually sank, and died a month later.

[1] Mackenzie, J., *Diseases of the Heart*, third edition, Lond., 1913, Appendix, Case 64.

[2] *Op. cit.*, Appendix, Case 65.

[3] *Op. cit.*, Appendix, Case 66.

Case XXXVIII., recorded by Mackenzie.[1]—Male, born in 1833, had enjoyed good health until 1901, when he began to have attacks of loss of consciousness. As a rule, these attacks came on suddenly; he would fall and immediately recover, but would feel dazed and weak. Once he lay unconscious for several hours, with a pulse so small that the doctor in attendance thought he was dead on several occasions. About six years later a severe attack of flutter, with a pulse-rate of just over 200 per minute, occurred. He became very restless, and his mind wandered. No remedies were efficacious. During the succeeding month the pulse-rate usually varied in rate from 74 to 180—being sometimes regular, at other times irregular. Slight excitement or exertion would often induce an attack of very rapid heart action, and the patient gradually became weaker and died.

Case XXXIX., recorded by Mackenzie.[2]—Paroxysmal tachycardia, probably due to auricular flutter. Male, aged 27, had suffered from rheumatic fever on two occasions. The paroxysmal attacks used to begin suddenly. At first they were infrequent and lasted for only a few minutes, but subsequently they occurred every few weeks and usually lasted from half an hour to six hours. If he took ipecacuanha wine the attacks usually ceased after he was sick and vomited. During the course of a third attack of rheumatic fever, a paroxysmal attack started and persisted for five days. This attack promptly ceased, and the patient experienced great relief after vomiting had been induced by ipecacuanha. Subsequently he said he could stop an attack by swallowing and belching air. This procedure was found to induce a slight and transient degree of heart-block. The patient gradually improved, and for the last two years he has been able to follow his profession, though he is occasionally liable to be crippled by a short attack.

Case XL., recorded by Mackenzie.[3]—Female, aged 16, had suffered from rheumatic fever, and for some years she had complained of breathlessness and palpitation on exertion. The records sometimes showed the characteristic features of auricular fibrillation; at other times there was a rapid regular rhythm suggestive of auricular flutter.

[1] Mackenzie, J., *Diseases of the Heart*, third edition, Lond., 1913, Appendix Case 67.

[2] *Op. cit.*, Appendix, Case 68.

[3] *Op. cit.*, Appendix, Case 69.

Case XLI., recorded by Fulton.[1] — The patient was a jeweller, aged 48, suffering from progressive chronic nephritis. For a year before admission to hospital he had been breathless on exertion, and for five weeks he had been dropsical. After he had been in hospital for three months, he suddenly developed an irregular action of the heart which persisted for a month. The return to the normal rhythm was sudden. There were no special symptoms during the attack, except that the œdema was more marked, and towards the end of the attack Cheyne-Stokes breathing developed. Digitalis was administered during the greater part of the attack, and meanwhile the auricles were probably fluttering at a rate of 286 to 308 per minute. At first the ventricles responded to each alternate auricular beat. Subsequently the ventricular responses were irregular, but there was definite and uniform grouping of the ventricular beats, similar to those in Fig. 104.

Case XLII., recorded by Fulton.[1]—A man, aged 54, with gradually increasing dyspnœa and œdema, came under observation with chronic nephritis and an enlarged heart. His pulse, at a rate of 135 to 138 and with marked alternation, was not influenced by posture or exercise. In the venous tracing the rate of the auricular waves was 272 per minute. Rest in bed induced some improvement in the patient's general condition, but the pulse-rate was not retarded until digipuratum had been given for eight days. The pulse then became slower and irregular in consequence of impaired and irregular conduction of stimuli to the ventricles. About one month later, the patient was again suffering from œdema and paroxysmal attacks of dyspnœa, and a fortnight later the auricles were in fibrillation.

Case XLIII., recorded by Hume.[2]—A girl, aged 7 years, was admitted to hospital on the fourth day after the onset of diphtheria. On the ninth day of illness auricular extrasystoles were recorded. On the tenth and eleventh days, when there were evident signs of collapse, the heart was beating with a nodal rhythm. On the twelfth day the extremities were cold, the child was apathetic and pallid, the pulse-rate varied from 90 to 102, and the auricles were probably in flutter. The child died three days later. At the post-mortem examination the

[1] Fulton, F. T., "'Auricular Flutter,' with a Report of Two Cases," *Archives of Intern. Med.*, Chicago, 1913, xii., 475.

[2] Hume, W. E., "A Polygraphic Study of Four Cases of Diphtheria, with a Pathological Examination of Three Cases," *Heart*, Lond., 1913-14, v., 25.

lungs presented patches of broncho-pneumonia and of collapse; the kidneys presented acute nephritis. The heart-muscle was pale and friable; the valves were healthy. Both the auricular and ventricular musculature showed fatty degeneration, and in the ventricles there was also a gross degree of interstitial myocarditis. The atrio-ventricular node and bundle appeared to be perfectly healthy.

Case XLIV., recorded by Hume,[1] was a boy, aged 5 years, who was suffering from diphtheria. A nodal rhythm was recorded on the eighth day of illness. Two days later, while the nodal rhythm persisted, signs of collapse ensued. Auricular flutter, at a rate of about 500 per minute, and with a ventricular rate of 96 to 132 per minute, probably supervened on the eleventh day, two days before the child died. The musculature of the auricles and ventricles presented fatty degeneration; the sinus node was inflamed; the atrio-ventricular node was slightly congested, but the bundle and its branches were healthy (see also p. 33).

Cases XLV. and XLVI.[2]—Fahrenkamp examined a series of cases of "arhythmia perpetua," and in two instances (Nos. 30 and 33 of his series) there was a rhythmic auricular tachysystole. The first case was a man, aged 50, affected with mitral stenosis and incompetence in whom the auricular rate was 240 to 350 (usually 300), and the ventricular rate 58-82 per minute. The second case was a man, aged 47, suffering from myocarditis, mitral stenosis, and mitral incompetence. The auricular beats were rhythmic at a rate of 190 to 240 per minute, whereas the ventricles were beating irregularly at a rate of only 50-60 per minute. Slight exertion induced a paroxysm in which the ventricular rate attained the same frequency as that of the auricles.

Fahrenkamp tabulates thirty-six other cases in which ventricular irregularity was associated with "arhythmic auricular tachysystole" at a rate of about 300-400 per minute. In these cases the auricular deflexions were irregular in rhythm, form, and amplitude, and may be regarded as indicating coarse fibrillation rather than auricular flutter.

Case XLVII.[3]—A customs officer, aged 52, who had been a temper-

[1] Hume, W. E., "A Polygraphic Study of Four Cases of Diphtheria, with a Pathological Examination of Three Cases," *Heart*, Lond., 1913-14, v., 25.

[2] Fahrenkamp, K., "Ueber das Elektrokardiogramm der Arhythmia perpetua," *Deutsch. Arch. f. klin. Med.*, Leipz., 1913, cxii., 302.

[3] Mathewson, G. D., "A Case of Auricular Flutter," *Edin. Med. Journ.*, Edin., 1913 (N. S.), xi., 500.

ate man and had never been ill except for one attack of acute rheumatism at the age of 26, had been complaining for eight months of pain in the region of the stomach, and for two months of dyspnœa. On rising in the morning he had often suffered from palpitation and giddiness. He had remained at work, however, until he was admitted to the Edinburgh Royal Infirmary.

His heart was dilated, but no murmurs were audible; there was no œdema of the feet or lungs, and no albuminuria. The auricles were fluttering at a rate of 298 to 312 per minute. The ventricular rate (156 to 164) was at first one-half of the auricular rate, but under treatment with digitalis the ventricular responses began to vary from 2 : 1 to 3 : 1, and thereafter a continuous 3 : 1 rhythm was established. On the ninth day of treatment, the auricles passed into fibrillation, with an irregular pulse at a rate of 60-80 per minute. This persisted for four weeks, and during the whole of this period, although no digitalis was being given, the patient felt well.

At the end of one month the auricular flutter, at a rate of 298-312 per minute, reappeared suddenly with symptoms of giddiness and faintness, and with a ventricular rate one-half that of the auricles. Digitalis had the same effect as before in slowing the ventricles, and ultimately in producing auricular fibrillation. This persisted until the patient left hospital, his general condition being then greatly improved.

Cowan[1] records three cases, and refers to four others in which the polygraphic records seemed clearly to prove flutter.

Case XLVIII. (Cowan's first case).—A shipyard labourer, aged 51, had been suffering for one year from paroxysms of palpitation, giddiness and shortness of breath, and from a gnawing pain in the epigastrium for three weeks. He became very weak and ill. There was no œdema, but the liver was large and somewhat tender, and the urine contained albumin. The area of cardiac dulness measured 5½ inches transversely; the sounds were muffled and indistinct, but pure. At the outset the ventricular action, at a rate of 136 to 150, was extremely irregular, series of 5 to 20 beats of uniform rhythm and force being separated from one another by pauses of longer duration, or by a number of beats at a less frequent rate. The pulse-rate fell suddenly and the rhythm became perfectly regular. The patient was conscious of the improvement and of the cessation of the flutter. His subsequent progress was good; the

[1] Cowan, J., *Diseases of the Heart*, Lond., 1914, chap. xvi.

area of cardiac dulness rapidly diminished to 4¼ inches, and the hepatic engorgement and pain passed away.

Case XLIX.—The second case recorded by Cowan[1] was a wire-drawer, aged 61, who had been a healthy man save for a brief attack of dropsy three years previously. For about four months he had been complaining of nausea, giddiness, cough, pain in the chest, and paroxysms of nocturnal dyspnœa. On admission to hospital he was dropsical; there was diffuse bronchitis, the liver was enlarged, double aortic murmurs were audible, and the Wassermann test was positive. About six weeks later his condition became worse. His ventricular action at a rate of 140 to 178 per minute was perfectly rhythmic, and the diastolic murmur disappeared. The ventricular rate, after remaining very frequent for eleven days, fell suddenly to 92, and afterwards to 76. The rhythm was then notably irregular, and the auricles were in fibrillation. The patient had been taking digitalis meanwhile. On the following day the frequent cardiac action (140 to 174 per minute) recurred and continued for eighteen hours, when the rate fell suddenly to 88. The rhythm from this time onwards remained wholly regular, except for an occasional extrasystole.

Case L.—The third case of Cowan's[1] series was a lad, aged 17, who complained of attacks of palpitation, which he dated from a fright that he had received at the age of twelve. For two months the attacks had been more frequent; he had become breathless on exertion, and easily tired. The pulse-rate varied from 60 to 214. The paroxysms, which began and ended abruptly, might pass off in a few minutes or might last for twenty-four hours. When the paroxysms ceased the enlargement of the right heart and of the liver speedily disappeared. The pulse-rate was always increased by examination, and lessened during sleep. Digitalis, opium, belladonna, and compression of the vagus seemed to have little influence on the rate of the heart.

Case LI. (recorded by Hoffmann).[2]—A somewhat delicate boy suffered from acute gastro-enteritis at the age of ten, and thereafter he began to complain of cardiac symptoms. He remained in fairly good health, however, until, at the age of fourteen, his heart's action became

[1] Cowan, J., *Diseases of the Heart*, Lond., 1914, 205, 207.

[2] Hoffmann, A., *Die Elektrographie als Untersuchungsmethode des Herzens und ihre Ergebnisse*, Wiesbaden, 1914, 188-193.

strikingly irregular after a slight follicular tonsillitis. The heart was small, the sounds were pure, the pulse soft and wholly irregular at a rate of about 120 per minute. The auricular rate was about 280 per minute, and the conduction of stimuli to the ventricles was sometimes notably depressed. The patient was under observation for three months, and during the whole of this period the flutter persisted.

Case LII.[1]—A man, aged 66, had an attack of paroxysmal tachycardia thirty-two years previously. The attack was brought on by severe strain, and lasted for several hours. Thereafter he had occasional attacks, especially after drinking cold water or eating indigestible food. The attacks began and ended suddenly, and usually lasted for several hours. During the attacks the pulse-rate often rose to 140-160, and occasionally to 180 per minute. Even during the attacks he had no dyspnœa, but of late he had complained of sub-sternal pain. The heart was not enlarged; the aortic curve was prominent; the sounds were pure. The auricles were beating at a rate of 300 per minute, with an auriculo-ventricular ratio of 2 : 1 or 3 : 1. Compression of the left vagus produced transient slowing of the ventricles only.

Case LIII.[1]—A woman, aged 65, had been subject to slight palpitation since her youth, but the attacks had been worse since the age of sixty. The heart was slightly enlarged to the left, the sounds were pure, but the second sound at the aortic area was somewhat accentuated. The pulse-rate was 164-176, the rhythm was regular, and the auricles were beating at twice the rate of the ventricles. Compression of the vagus led to slowing of the ventricles, and the true nature of the auricular action was thereby revealed. Subsequently the patient became dropsical, and she died suddenly.

Digitalis was administered to the three cases recorded by Hoffmann, but the drug did not influence the ventricles, and auricular fibrillation did not supervene.

[1] Hoffmann, A., *Die Elektrographie als Untersuchungsmethode des Herzens und ihre Ergebnisse*, Wiesbaden, 1914, 193-202.

CHAPTER V

CLINICAL FEATURES

Even in an apparently healthy heart the onset of auricular flutter, with pronounced ventricular acceleration, greatly impairs the efficiency of the organ, and, as a rule, causes distressing, or even urgent, symptoms. In other instances the myocardium is known to be diseased, and grave indications of heart failure have been recognised prior to the onset of auricular flutter, yet when this disorder does ensue all the patient's symptoms become aggravated.

The symptoms specially associated with auricular flutter are not direct manifestations of the extreme acceleration of the auricles, but are due to ventricular acceleration and to secondary alterations in the arterial and venous pressures. In a normal heart beating at a rate of 75 per minute the duration of ventricular systole is about 0·30 second, and that of ventricular diastole about 0·5 second. When the auricles are in flutter the rate of their contraction is usually about 250-300 per minute, whereas the ventricular rate is usually one-half of the auricular rate. In auricular flutter, as in other forms of tachycardia, the ventricular acceleration occurs mainly at the expense of diastole, although the systolic phase may also be curtailed somewhat. For example, in Case II., with auricular and ventricular rates of 283 and 141·5 per minute respectively, ventricular systole lasted about 0·245 second, whereas ventricular diastole lasted only 0·175 second. In Case III. the duration of ventricular diastole was reduced to about 0·14 second.

The curtailment of ventricular diastole to one-third, or less, of its normal duration does not necessarily imply imperfect filling of the ventricles, for we know that in health filling is almost maximal at an early period of diastole. The curtailment of diastole, however, entails a period of rest shorter than normal, and the ventricles are again stimulated to contract at a time when their contractility is yet only partially restored. The ventricular beats are therefore enfeebled, the arterial pressure must necessarily fall, and the circulation of blood become impaired in the systemic and pulmonary circulations.

Moreover, from the cyanosis, œdema, dropsy, and other clinical manifestations, we may conclude that there is at the same time a rise of venous pressure.

In considering the clinical features of auricular flutter it is convenient to recognise four groups of cases—(1) Auricular flutter in an apparently healthy heart; (2) auricular flutter supervening during the course of chronic cardiac disease; (3) auricular flutter in association with partial heart-block; and (4) auricular flutter with complete heart-block. Flutter comes on in the form of paroxysmal attacks which may subside in the course of a few minutes, or may persist for days, months, or even years.

I. Auricular Flutter in an Apparently Healthy Heart

In an individual who had previously enjoyed good health, a paroxysmal attack of flutter may be excited suddenly, and without any warning, under the influence of emotion or physical exertion, or the attack may apparently start during sleep, the patient awaking to find himself ill and his pulse greatly accelerated. In some instances it may be possible to refer the onset of the attack to some acute infective disease or toxæmia, such as influenza or gastro-intestinal catarrh.

Premonitory symptoms, due mainly to extrasystoles, may have occurred now and again for many months or years. There may have been vague and momentary sensations of discomfort in the precordial region, such as that of "a flapping in the chest" or of "the heart standing still," indicating an occasional extrasystole. Again, the occurrence of multiple extrasystoles may have given rise to the alarming symptom of "tremor cordis" so well described by Balfour[1] and Gibson.[2] In this condition the patient complains of "fluttering in the chest," of "fluttering of the heart," or, as Gibson says, "the individual affected feels as if he had a timid bird fluttering within the bosom." The rapid, feeble, and irregular beats of the ventricles in "tremor cordis" are recognisable by the hand laid on the precordia, as well as by the ear. The attack may last for a few seconds, and usually ends abruptly with a pause and then an unduly forcible beat.

The symptoms of auricular flutter are essentially due to the great acceleration of the ventricles under the influence of the fluttering

[1] Balfour, G. W., *Clinical Lectures on Diseases of the Heart and Aorta*, Lond., 1898, 282.

[2] Gibson, G. A., *The Nervous Affections of the Heart*, Edin. and Lond., 1904, 92, 93.

auricles. In most cases the initial symptom of each paroxysm is palpitation, which is often very distressing. This symptom usually persists while the ventricular rate remains accelerated, and if the attack be a prolonged one the patient often complains of dyspnœa, precordial pain, weakness, and prostration. In some instances vertigo ensues as a result of the weak and extremely rapid ventricular action, and there may be occasional syncopal attacks. These are most apt to ensue when the ventricles respond to each auricular beat and when the ventricular rate therefore rises to about 200 to 300 per minute.

During an attack of auricular flutter the heart-sounds acquire the fœtal rhythm, and if the attack be prolonged for some weeks a mitral systolic murmur may develop in consequence of cardiac dilatation.

In some instances, however, even when the ventricular rate exceeds 140 or 150 beats per minute, the patient experiences wonderfully little discomfort beyond slight palpitation, and he may continue to lead an active life for several days, or even weeks, after the paroxysm of auricular flutter began. In such cases the symptoms may gradually become more urgent the longer the flutter lasts. In other instances the patient may remain remarkably free from all urgent symptoms for many months, provided he leads a quiet life. Case XXI. affords a good example.

When the auricles are in flutter the ventricular rate is usually one-half of the auricular rate. The latter is most frequently between 250 and 300, but may attain, or even exceed, a rate of 370 per minute. In most instances the ventricular rate is therefore between 140 and 150 per minute, nevertheless a rate of 160 is not uncommon.

When the rate of the ventricles is constantly one-half of the auricular rate, the ventricular beats and the arterial pulse, although greatly accelerated, are rhythmic. The pulse is of small volume and may be alternating if the rate exceeds 150 per minute. The pulse is sometimes so rapid and feeble as to be almost imperceptible at the wrist.

Less frequently the ventricular rate is constantly one-third of the auricular. Under these circumstances there is probably defective conduction in the atrio-ventricular bundle. Case XXX., with an initial ventricular rate of only 87 per minute, may be taken as an example. In cases of this nature the auricular flutter is particularly apt to escape recognition, because the pulse is rhythmic and not notably accelerated.

In other instances, when the ratio of auricular to ventricular systole varies from 2:1 to 3:1, from 2:1 to 4:1, or from 3:1 to 4:1, the irregularity of the cardiac impulse and arterial pulse is a striking feature

which attracts the attention of the physician. Lastly, there may be paroxysmal attacks in which the arterial pulse, according to Mackenzie, attains a rate of 290-300 per minute, and in Case XXXVIII. a rate of just over 200 was attained. In such instances the auriculo-ventricular ratio is 1 : 1 instead of 2 : 1, as is usually the case during the initial stage of auricular flutter.

The duration of the paroxysmal attacks varies widely in different patients and in different attacks in the same patient. An attack may last for only a few minutes, for some hours, or for several days or weeks. Initial attacks are usually of shorter duration than subsequent ones. They may pass off spontaneously. An attack which has persisted for a day or two and caused the patient much anxiety and distress may terminate while he is asleep. When he awakens, he at once feels that he is better. The palpitation, dyspnœa, and precordial discomfort have vanished, his pulse-rate has fallen from 150 or 160 to about 80 per minute, and in the course of a few days the patient is able to get out of bed, and may even be able to resume his former mode of life without experiencing any uneasiness or distress. Once an attack of flutter has occurred there is a distinct tendency for attacks to recur from time to time.

II. Auricular Flutter Supervening during the Course of Chronic Heart Disease

The majority of cases of auricular flutter are included in this group. The patient's history and physical condition indicate that he has been affected for many years with mitral disease or with arterio-sclerosis and chronic interstitial myocarditis. There may be co-existent chronic nephritis (Cases XLI. and XLII.). Aortic lesions are seldom demonstrable.

In some instances the patient, although affected with chronic valvular or myocardial disease, has been able to follow his ordinary mode of life until with the onset of flutter there arise, suddenly or insidiously, symptoms such as palpitation, dyspnœa, precordial pain, insomnia, and prostration. Sudden attacks of faintness or of complete loss of consciousness may occur from time to time if the ventricular acceleration becomes extreme (Cases VI., XXXVIII.). Bodily fatigue, excessive strain, worry, or emotion, may be the immediate cause that excites an attack of flutter, or it may arise during some acute intercurrent affection such as bronchitis, intestinal catarrh, or influenza.

On the onset of flutter, the patient's condition almost invariably

becomes worse, much in the same way as so frequently occurs on the onset of auricular fibrillation. The change may develop suddenly or gradually. For a few days the auricles may be fluttering and the arterial pulse may be so rapid and feeble as to be almost imperceptible at the wrist, and yet the patient may be conscious of no particular symptoms except palpitation and some increase of dyspnœa. Within the course of a few days at most the symptoms become more urgent. The patient is usually most comfortable when he lies propped up in bed. The face acquires a dusky tint, the liver becomes congested and enlarged, tenderness is often developed on deep pressure over this organ in the right hypochondrium or epigastrium, crepitations appear at the bases of the lungs, the feet may become œdematous, the complexion jaundiced, and the urine scanty and albuminous. If the flutter persists and the ventricular rate remains constantly rapid, somnolence, stupor, and Cheyne-Stokes respiration may develop. In other instances the patient becomes restless or even mildly delirious.

The rate of the ventricles and of the arterial pulse depends on the rate at which the auricles are fluttering and on the ratio of auricular to ventricular systole (2 : 1, 3 : 1, or 4 : 1), as already described. In most cases there is an initial ratio of 2 : 1, and the ventricles beat rhythmically at a rate of about 125-160 per minute. In Cases XVIII., XXXVIII., and XLVI. the ratio was occasionally 1 : 1, and in Case XVIII. the ventricular rate rose to 290-300 per minute. Another patient, aged 37, whom I saw with Dr. Edwards of Bridge of Earn, was suffering from mitral stenosis and from auricular flutter that had come on abruptly and had persisted almost continuously for five months. When the patient was recumbent the heart was beating with a normal rhythm and at a rate of 125 per minute, but when she sat up in bed the rate of the auricles and the ventricles, as recorded graphically, rose to 255 per minute.

When auricular flutter supervenes in a case of mitral disease all the antecedent endocardial murmurs usually become obscured owing to the extreme acceleration of the ventricles. An antecedent mitral presystolic murmur will almost certainly disappear. Indeed, it is almost impossible to hear any diastolic murmur, or to time it even if it be audible, when the ventricular diastole is curtailed to 0·14 second, as is the case when the ventricular rate is about 150 or 160 per minute. But even if the ventricular rate be not greatly accelerated, as when it has fallen to one-fourth of the auricular rate, a genuine presystolic murmur can hardly arise, because when the auricles are fluttering there

will be no notable rise of intra-auricular pressure during the last phase of ventricular diastole such as occurs when the auricles contract once at their normal time in the cardiac cycle.

Auricular flutter, with a pulse-rate of about 150, may alternate from hour to hour, or from day to day, with a physiological rhythm in which the rate is about 70-80 per minute, or the flutter may persist for days or for weeks. In many instances the flutter ultimately passes into auricular fibrillation, but before the latter is finally established there may be many transitions from flutter to fibrillation and *vice versâ*, or again between flutter, fibrillation, and a normal rhythm. During these transitional phases it may be difficult to determine whether the auricles are in flutter or in fibrillation, especially if electrocardiograms cannot be obtained or if, in the absence of any long ventriculo-diastolic pauses, satisfactory tracings of the auricular waves in the jugular pulse cannot be secured.

After persisting for several hours, days, or weeks, the flutter may subside, and the heart regain its physiological rhythm at a rate of about 70-80 per minute. In all likelihood the patient's general condition will then begin to improve speedily.

In other instances, even although the auricles continue in flutter, improvement begins whenever the ventricular rate diminishes, as often occurs under the influence of digitalis. If the patients continue to take this drug in doses sufficient to hold the ventricular action in check, they may be able to lead a quiet and fairly comfortable existence for many years. In such cases, however, there is always a risk of sudden death.

Again, auricular flutter may supervene as a terminal event after the patient has been confined to bed, cyanosed, breathless and dropsical, for many weeks or months. In Case III., for instance, the patient had been suffering for about nine months from heart failure consequent on mitral disease, and auricular flutter at a rate of 320 per minute set in ten days before his death. In this case the flutter was apparently due to acute pericarditis and sub-acute mitral endocarditis spreading into the auricular musculature. In Case VII., one of exophthalmic goitre and dilatation of the heart, the auricles probably passed into flutter three days before death.

III. Auricular Flutter with Partial Heart-block

The main clinical features are similar to those described in the preceding group of cases. Owing to the existence of partial heart-block,

however, the rate of the ventricles is less accelerated, and their rhythm is as a rule irregular.

In the two preceding groups we have been considering cases in which the ventricles, at the onset of auricular flutter, usually responded to every second auricular beat, so that there was, for example, a ratio of As : Vs :: 280 : 140. In those cases the transmission of every alternate stimulus to the ventricles was undoubtedly blocked. This blocking, which is probably due to a functional inability on the part of the atrio-ventricular bundle to transmit onwards stimuli at such a frequent rate, is an example of a natural protective mechanism. That it is not due to any pre-existing defect in the conducting system, as Lewis[1] maintains, is indicated by the fact that when the auricles of a healthy mammalian heart are set into flutter experimentally every alternate stimulus to the ventricles is likewise blocked.

There are a few cases of auricular flutter, however, in which there was apparently a pre-existing pathological depression of conductivity in the atrio-ventricular bundle whereby the ventricles did not receive each alternate stimulus from the auricles, but merely every third, fourth, or sixth stimulus. When the auriculo-ventricular ratio was 4 : 1 the ventricular rate was notably infrequent and the rhythm was liable to be irregular. The defect in conductivity may have been latent until it was revealed in consequence of the auricular acceleration.

The case recorded by Gibson[2] (Case XII.) is a beautiful illustration of auricular flutter in association with partial heart-block. When the auricles were beating rhythmically at a rate of 90 per minute, an occasional stimulus to the ventricles was blocked. When the auricles were fluttering at a rate of 196-200 per minute, the rate of the ventricles was usually one-fourth that of the auricles, and the ventricular rhythm was irregular. After death the atrio-ventricular bundle "showed a very considerable increase of fibrous tissue with wide separation of the muscle fibres constituting it."

The case recorded by Hertz and Goodhart[3] (Case XIV.) may also be referred to here. In Fig. 1 of their paper, the ventricular beats are coupled regularly at a rate of 80 per minute. The second beat of each

[1] Lewis, T., "Observations upon a Curious and not uncommon Form of Extreme Acceleration of the Auricle. 'Auricular Flutter,'" *Heart*, Lond., 1912-13, iv., 171.

[2] Gibson, G. A., "A Discussion on some Aspects of Heart-block," *Brit. Med. Journ.*, Lond., 1906, ii., 1113.

[3] Hertz, A. F., and Goodhart, G. W., "The Speed-limit of the Human Heart," *Quart. Journ. of Med.*, Oxford, 1908-9, ii., 213.

couple is so premature as to give the impression of being a ventricular extrasystole. Consequently, the ventricles responded to supra-ventricular stimuli only 40 times per minute, and as the auricular rate was 240 per minute there was a ratio of As : Vs :: 6 : 1, which suggests that the conductivity of the atrio-ventricular bundle was probably defective. The defect may have been due to vagus stimulation or to drug administration, however, and not to structural changes in the atrio-ventricular bundle, for the ventricular rate rose to 170 after an injection of $\frac{1}{50}$ grain of atropin.

IV. Auricular Flutter in Association with Complete Atrio-ventricular Heart-block

Whenever there is a lesion severing completely the main stem of the atrio-ventricular bundle the ventricular rhythm becomes wholly dissociated from that of the auricles. The ventricles beat with an independent rhythm in response to stimuli coming from the atrio-ventricular bundle below the lesion. The auricular beats may be slow or fast, regular or irregular, strong or weak, yet the rate, rhythm, and strength of the ventricular beats are wholly independent of the auricles. Moreover, the vagus and sympathetic influences that retard or accelerate the auricles in no wise affect the ventricles. The ventricular rate is slow, usually 30-36 per minute, the rhythm is usually absolutely regular, and the contractions are not necessarily enfeebled. Numerous cases have been reported in which, during life, exact records of complete atrio-ventricular dissociation were obtained, and post-mortem the main stem of the bundle was found to be severed.

One notable case of this nature was recorded by Gibson and myself[1] in 1909. In another case that had been under my own observation and in four similar cases recorded by P. Macdiarmid,[2] I found the main stem of the atrio-ventricular bundle severed by chronic inflammatory lesions.

Experimental and clinical observations indicate that unless the heart is under the influence of vagus stimulation, partial severance of the bundle does not cause complete heart-block. On the other hand, the block may have been complete during life, and yet no lesion can be discovered in the bundle. Only one case of this nature (Fig. 73)

[1] Gibson, G. A., and Ritchie, W. T., "A Historic Instance of the Adams-Stokes Syndrome due to Heart-block," *Edin. Méd. Journ.*, 1909 (N.S.) ii., 315, 507.

[2] Macdiarmid, P., *Heart-block and Adams-Stokes Disease*, Thesis, University of Edin., 1911.

has come under my notice. After a typical Adams-Stokes seizure in a man aged 75, the block remained complete for the two and a half months during which the patient was under observation, and it was not relieved by atropin. A fortnight after the patient was last seen, he died suddenly. Both nodes, the bundle, and the initial parts of both its branches, were examined in serial sections, but no lesion could be detected. The true cause of the heart-block was not elucidated until Miss Meiklejohn, to whom I am indebted for the examination of this heart, made serial sections of the inter-ventricular septum below the level of the pars membranacea septi. It was then found that both

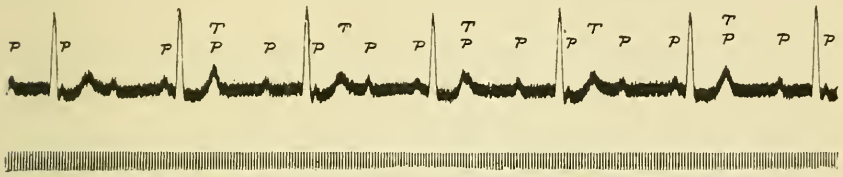


FIG. 73.—Complete heart-block, with an auricular rate of 97 and a ventricular rate of 38 per minute. Derivation I. 1 cm. = 1 millivolt.

branches of the bundle were almost wholly obliterated by dense fibrous tissue.

The only recorded instance of auricular flutter in association with complete heart-block is Case I. (p. 35). Antecedent to the onset of flutter there was complete heart-block, with auricular and ventricular rates respectively of 50-60 and 31-35 per minute. The onset of auricular flutter at a rate of 273 per minute did not disturb the rate, rhythm, or strength of the ventricular beats, and the patient was unaware of any fresh symptoms. This case demonstrates clearly that the symptoms associated with auricular flutter are not due to the flutter directly, but arise indirectly as a result of the great ventricular acceleration in response to the flutter.

CHAPTER VI

GRAPHIC RECORDS

When the auricles are in flutter, their contractions can often be recorded graphically. As characteristic tracings and electrocardiograms have already been considered in Chapter IV., only the salient features need be referred to here.

(1) *Sphygmograms.*—The rate and rhythm of the arterial pulse vary greatly in different cases and at different times in any one case. When the ventricular rate is constantly one-half of the auricular rate, the record is that of a rhythmic rapid pulse at a rate of about 130-

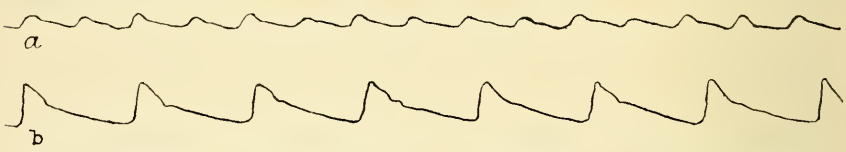


Fig. 74.—Sphygmograms in auricular flutter (Case II.). In *a* the ventricles are responding to every second auricular beat, and the pulse is frequent and alternating. In *b* the ventricles are responding to every fourth auricular beat.

160 per minute (Fig. 74, *a*). The pulse is often alternating and may be hyperdicrotic.

When the ventricles respond uniformly to every third or every fourth auricular beat, the arterial tracings do not necessarily differ in any essential respect from the normal (Fig. 74, *b*), although when the auriculo-ventricular ratio is 3 : 1 the pulse may be alternating, as in Case XXXII.

If the ventricles are responding irregularly to fluttering auricles, the arterial pulse tracing is at first sight wholly irregular. But when the tracing is analysed, it will usually be found that the pulse-beats occur in groups of uniform duration, representing multiples of the inter-auricular period. This is illustrated in Fig. 75. These records were obtained from a ploughman, aged 39, who had been suffering for four months from breathlessness and weakness, and who had been unfit for any work for three weeks. No history of acute rheumatism or syphilis could be elicited. When admitted to the Royal Infirmary under the care of Dr. Lovell Gulland, the patient was greatly troubled

with breathlessness and cough, the face was dusky with an icteric tint, the feet and lungs were œdematous, the liver was enlarged, and pressure over it elicited tenderness. The heart was moderately enlarged, but no murmurs were audible. The ventricles were beating at a rate of 140-160 per minute; the rhythm was usually irregular, as in Fig. 75, but

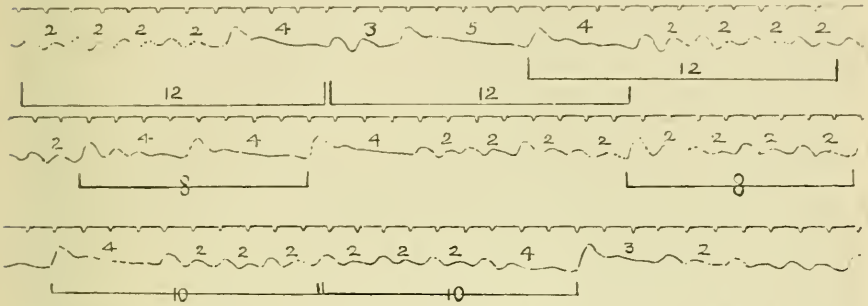


FIG. 75.—Sphygmograms from a case of auricular flutter with irregular ventricular responses and grouping of the arterial beats.

at times the pulse was rhythmic, and it was then alternating. The auricles were probably in flutter at a rate of about 320 per minute.

Digitalin was administered, and ten days later, when electrocardiograms could be recorded, the auricular deflexions, which were either irregular or regular at a rate of 380 per minute, represented either a coarse fibrillation or a form of auricular action with simultaneous flutter and fibrillation. One week later, when the ventricles were beat-

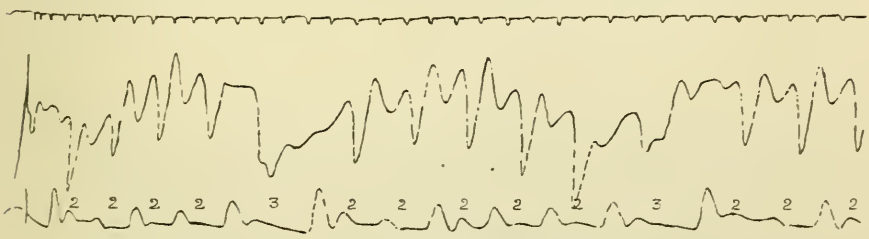


FIG. 76.—Auricular flutter. Jugulo-carotid and brachial tracings showing a ventricular response to every second or every third auricular beat.

ing slowly and irregularly, the auricular deflexions were of small size, irregular form and frequency, indicating auricular fibrillation. When the patient left hospital two months later, his general condition was greatly improved.

Another example of irregular ventricular responses in flutter is shown in Fig. 76 from Case II. The ventricles were usually responding to each alternate, but occasionally to every third, auricular beat. After the latter event there was a transient but well-marked pulsus

alternans. The irregularity of the pulse in this instance was therefore due to the ventricles responding irregularly to rhythmic auricular beats, and also to the delay of the smaller pulse waves when alternation developed. This delay can also be demonstrated by comparing the

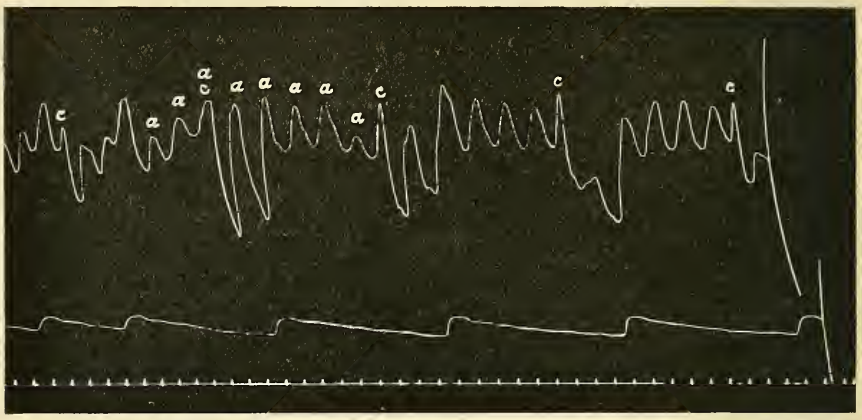


Fig. 77.—Jugulo-carotid and brachial tracings. Auricular flutter at a rate of 196 per minute. Successive ventricular beats are responses to every third, fifth, and sixth auricular beats. Case XII., 9th February 1906. The time record is 0·2 second.

interval between each deflexion R in an electrocardiogram with the corresponding pulse wave in a sphygmogram recorded simultaneously (Plate XXI., Fig. 106).

The irregularity of the arterial pulse may be intensified still further by ventricular beats failing to open the aortic valve and consequently

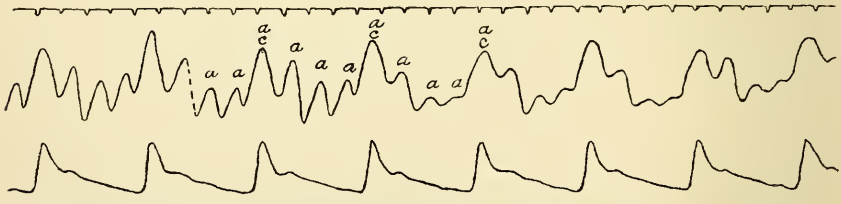


Fig. 78.—Auricular flutter at a rate of 266 per minute. The ventricles respond to every fourth auricular beat. Jugulo-carotid and brachial tracings (Case II.).

having no representative in the sphygmogram (Plate XXI., Fig. 106), and also by the occurrence of ventricular extrasystoles (Plate XV., Fig. 83).

2. *Jugulo-carotid Tracings.*—When the rate of the ventricles is infrequent and does not attain one-quarter of the auricular rate, the tracings usually present large, rhythmic auricular waves during each ventricular diastole (Figs. 31 and 77). During ventricular systole the auricular waves may be obscured by the waves c and v, but in some

PLATE XIV.

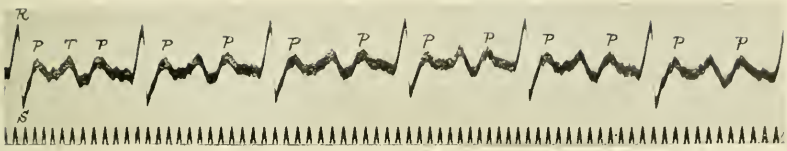


Fig. 79.—Auricular flutter. The ventricular rate is one-half that of the auricles. Case II. Derivation II. 1 cm. = 1 millivolt. (*Edin. Med. Journ.*, 1912, vol. ix.)

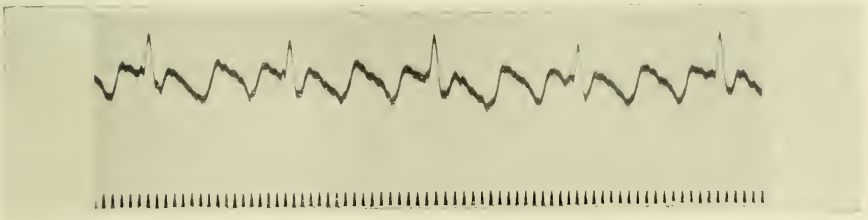


Fig. 80.—Auricular flutter. The ventricular rate is one-third that of the auricles. Case XLVII. Derivation II. 1·5 cm. = 1 millivolt. (G. D. Mathewson.) (*Edin. Med. Journ.*, 1913, vol. xi.)

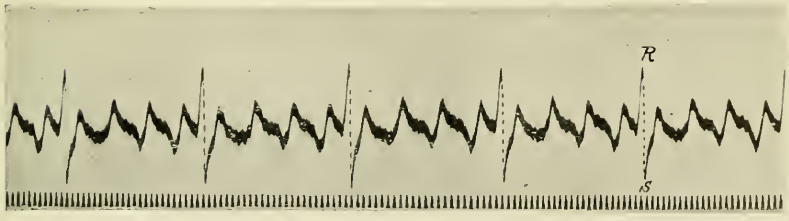


Fig. 81.—Auricular flutter. The ventricular rate is one-fourth that of the auricles. Case II. Derivation II. 1 cm. = 1 millivolt. (*Edin. Med. Journ.*, 1912, vol. ix.)

instances the auricular waves occurring during ventricular systole are larger than those during ventricular diastole (Fig. 77), because during the former phase the tricuspid orifice is closed and during the latter open.

If the auricles are beating uniformly four times as fast as the ventricles, the four auricular waves for each arterial pulse-beat (Fig. 78) must not be mistaken for *a*, *c*, *v*, and *h* waves of a normal rhythm (see p. 7). If the auriculo-ventricular ratio is constantly 3:1, the three waves in the jugular curve may simulate *a*, *c*, and *v* waves of a normal rhythm (see p. 112).

When the auricular rate is uniformly twice that of the ventricles, the jugular pulse is of the ventricular form—the curve rises with the onset of ventricular systole, and after a more or less well-marked systolic plateau, falls when the tricuspid valve opens (Figs. 43, 51, and 72).

If the ratio of As:Vs is irregular, the form of the jugulo-carotid tracing is inconstant, being of the ventricular form when the ventricles respond to every alternate auricular beat, and presenting definite auricular waves during each of the prolonged ventricular diastoles.

3. *Electrocardiograms.—The auricular deflexions* are rhythmic, and usually occur at a rate of about 200-320 per minute. The maximum rate in my own cases was 377 per minute (Case V.), but in one of Hume's cases (Case XLIV.) the rate was probably about 500 per minute. In each individual case the deflexions are not merely rhythmic, but as recorded by each derivation are almost invariably of constant form, except in so far as they may be distorted by ventricular deflexions. By derivation I. (right hand and left hand) the deflexions are monophasic and of comparatively small amplitude. They may indeed be so small as to be almost imperceptible, as in Case III. By derivations II. (right hand and left foot) and III. (left hand and left foot), they are larger and often diphasic (Plate IX.), with the initial deflexion upwards. The auricular beats usually succeed one another so rapidly that when one auricular deflexion ends another begins (Plate XIV.).

In their diphasic form the auricular deflexions representing flutter differ from the normal, and correspond with those that may be observed when the auricles are under vagus inhibition. This abnormal (diphasic) form probably signifies that the auricular contractions are initiated at some site other than the normal one.

If the auricular deflexions vary somewhat in form, size, and rhythm, and yet their rate does not exceed about 370 per minute, the auricular action is probably a combination of flutter and fibrillation (Fig. 83). In true fibrillation the auricular deflexions are small, irregular in size

and rhythm, and occur at a rate of usually 380-520 per minute. In Case V. gradual transitions from auricular fibrillation to flutter were recorded (see Plate XXI.), and similarly in the case described by Canby Robinson[1] flutter and fibrillation alternated with each other several times. In this case the auricles were fluttering at a rate of 380 per minute, while the ventricular beats were rhythmic at a rate of 95 per minute. The ventricular beats then became arrhythmic and no definite correlation could be determined between the auricular and the ventricular beats, probably because the auricular activity was a combination of flutter and fibrillation. A few minutes later the electrocardiogram was characteristic of auricular fibrillation.

The ventricular deflexions in cases of auricular flutter are of such form as indicates supra-ventricular stimulation. The amplitude of the individual deflexions *Q*, *R*, *S*, and *T* varies in different cases according to the nature of the valvular lesion and the predominance of hypertrophy in right or left ventricle. For example, in Case III., with mitral stenosis, *R* is larger by derivation III. than by derivation I., whereas *S* is larger by derivation I. than by derivation III. In Case I., however, with predominant hypertrophy of the left ventricle, *R* is larger by derivation I. than by derivation III., while *S* is larger by derivation III. than by derivation I. (see Plates IX. and XI.).

When the ventricular rate is extremely high, not only is ventricular diastole curtailed to about one-third of its normal, but the duration of ventricular systole is also shortened. Thus in Case II., with a ventricular rate of 141·5 per minute, ventricular systole lasted about 0·245 second as against the normal 0·30 second.

[1] Robinson, G. Canby, "The Relation of the Auricular Activity following Faradization of the Dog's Auricle to Abnormal Auricular Activity in Man," *Journ. of Exp. Med.*, New York, 1913, xviii., 704.

PLATE XV.

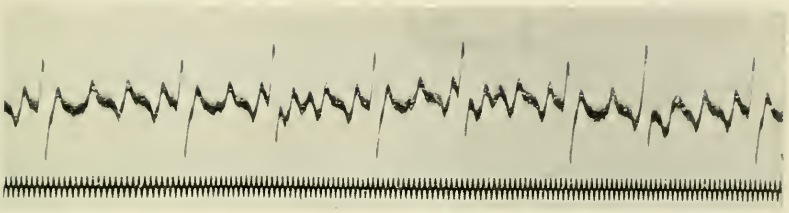


Fig. 82.—Auricular flutter at a rate of 276·4 per minute, with irregular ventricular responses. Case II. (22nd August 1912). Derivation II. 1 cm. = 1 millivolt.

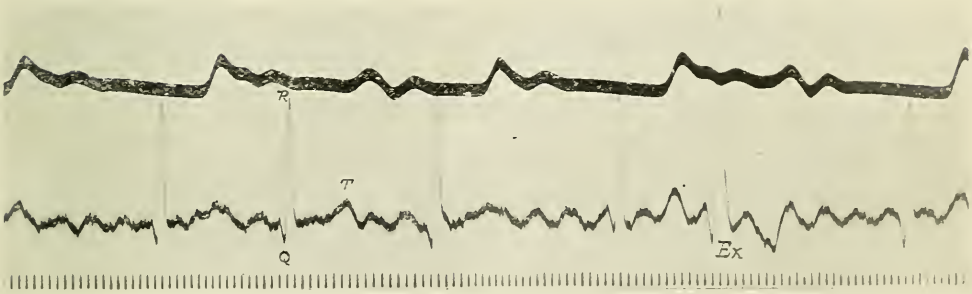


Fig. 83. —Auricular flutter and fibrillation combined. One ventricular beat (*Ex*) is an extrasystole. Brachial pulsations and electrocardiogram by derivation III. Case V. 1 cm. = 1 millivolt. (*Quart. Journ. Med.*, vol. vii.)

CHAPTER VII

THE ACTION OF THE VAGUS AND SYMPATHETIC

The rate of stimulus production, the excitability, contractility, conductivity, and tone of the heart, may be influenced by many factors, and notably by those of nervous origin.

Chronotropic influences are expressed by changes in the rate of stimulus production. This may be accelerated (positive chronotropic effect) or retarded (negative chronotropic effect). *Bathmotropic influences* are those upon the excitability of the heart muscle; positive bathmotropic influences increase, whereas negative bathmotropic influences lower, its excitability. *Dromotropic influences* are those upon conductivity within the heart, and are most evident upon the atrioventricular bundle system. Positive and negative dromotropic influences respectively improve and impair the conduction of stimuli. *Inotropic influences* are expressed by increase or decrease of contractility. They may be positive or negative according as the contraction of muscle fibres is strengthened or weakened. *Tonotropic influences* are those affecting the tone of the heart-muscle; when positive they heighten, when negative they lower the muscle tone.

The effects of each of these influences may be primary or secondary, and, as pointed out by Wenckebach,[1] they are often of complex nature. Thus a primary negative chronotropic influence lessening the rate of the auricles may cause secondary dromotropic and inotropic effects, as revealed by improved conduction of stimuli and stronger contractions. Again, a primary negative dromotropic influence on the auricles, causing blocking of stimuli within their walls, may cause a secondary negative inotropic effect, which is manifested by weakening of the beats.

In order to appreciate correctly the action of the vagus and sympathetic on the heart when the auricles are fluttering, it is necessary to understand the effects on hearts with a physiological rhythm.

[1] Wenckebach, K. F., *Die Arhythmie als Ausdruck bestimmter Funktionsstörungen des Herzens*, Leipz., 1903, 134.

I. The Action of the Vagus on the Heart Beating with a Physiological Rhythm

The striking effects of the vagus on the normal mammalian heart, as recorded by MacWilliam,[1] Roy and Adami,[2] Bayliss and Starling,[3] Gaskell,[4] Engelmann,[5] Hering,[6] Einthoven,[7] and other investigators, are well known. Recent experimental work on animals suggests that the right vagus exerts its influence especially on the sinus node and the auricles, whereas the left vagus has a preponderant influence on the atrio-ventricular conducting system and the ventricles, but it is doubtful whether this holds good for the human heart.

The vagus effects on the human heart, originally studied by Czermak,[8] have been described by Robinson and Draper[9] and by myself,[10] and have been shown to be comparable to those on the lower mammalian heart. Stimulation of the vagus—right or left—may be effected by means of digital compression upon the nerve in the neck, whereas the vagus endings in the heart can be paralysed by the subcutaneous injection of 0·02-0·03 grain of atropin sulphate given subcutaneously.

[1] MacWilliam, J. A., "On the Phenomena of Inhibition in the Mammalian Heart," *Journ. of Physiol.*, Camb., 1888, ix., 345.

[2] Roy, C. S., and Adami, J. G., "Contributions to the Physiology and Pathology of the Mammalian Heart," *Philos. Trans. Roy. Soc. Lond.*, B, 1893, clxxxiii., 199.

[3] Bayliss, W. M., and Starling, E. H., "On some Points in the Innervation of the Mammalian Heart," *Journ. of Physiol.*, Camb., 1892, xiii., 407.

[4] Gaskell, W. H., "On the Rhythm of the Heart of the Frog, and on the Nature of the Action of the Vagus Nerve," *Philos. Trans. Roy. Soc. Lond.*, 1882, clxxiii., 3, 993; "The Nature of the Action of the Cardiac Nerves," *Schäfer's Text-book of Physiology*, Edin. and Lond., 1900, ii., 203-221.

[5] Engelmann, Th. W., "Ueber die Wirkungen der Nerven auf das Herz," *Arch. f. Anat. u. Physiol.* (Physiol. Abt.), Leipz., 1900, 315.

[6] Hering, H. E., "Ueber die unmittelbare Wirkung des Accelerans und Vagus auf automatisch schlagende Abschnitte des Säugethierherzens," *Arch. f. d. ges. Physiol.*, Bonn, 1905, cviii., 281; "Experimentelle Studien an Säugethieren über das Elektrokardiogramm," *ibid.*, 1909, cxxvii., 155.

[7] Einthoven, W., and Wieringa, J. H., "Ungleichartige Vaguswirkungen auf das Herz, elektrokardiographisch untersucht," *Arch. f. d. ges. Physiol.*, Bonn, 1913, cxlix., 48.

[8] Czermak, J. N., "Ueber mechanische Reizung des Nervus vagus beim Menschen," *Gesammelte Schriften*, Leipz., 1879, i., 2, 779.

[9] Robinson, G. Canby, and Draper, G., "Studies with the Electrocardiograph on the Action of the Vagus Nerve on the Human Heart," *Journ. of Exper. Med.*, New York, 1911, xiv., 217.

[10] Ritchie, W. T., "The Action of the Vagus on the Human Heart," *Quart. Journ. of Med.*, Oxford, 1912-13, vi., 47.

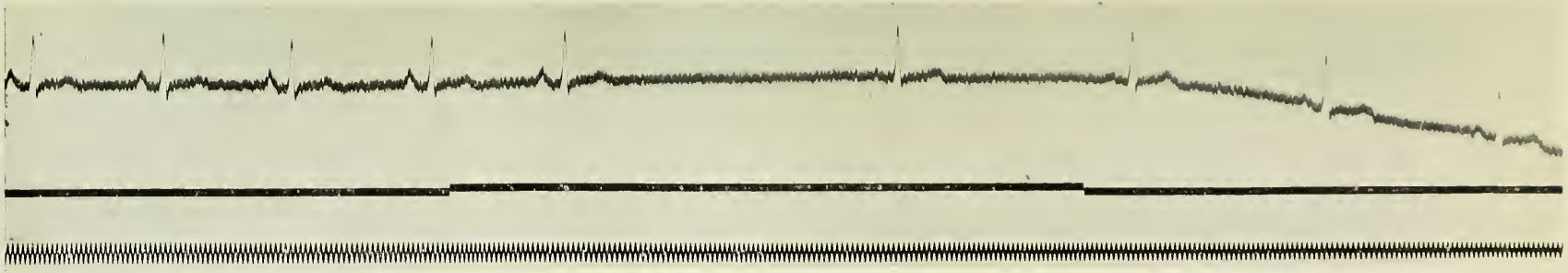


Fig. 84.—Compression of the right vagus during the period shown by the signal retards the whole heart and depresses auricular contractility. Derivation II. 1 cm. = 1 millivolt. (*Quart. Journ. Med.*, vol. vi.)

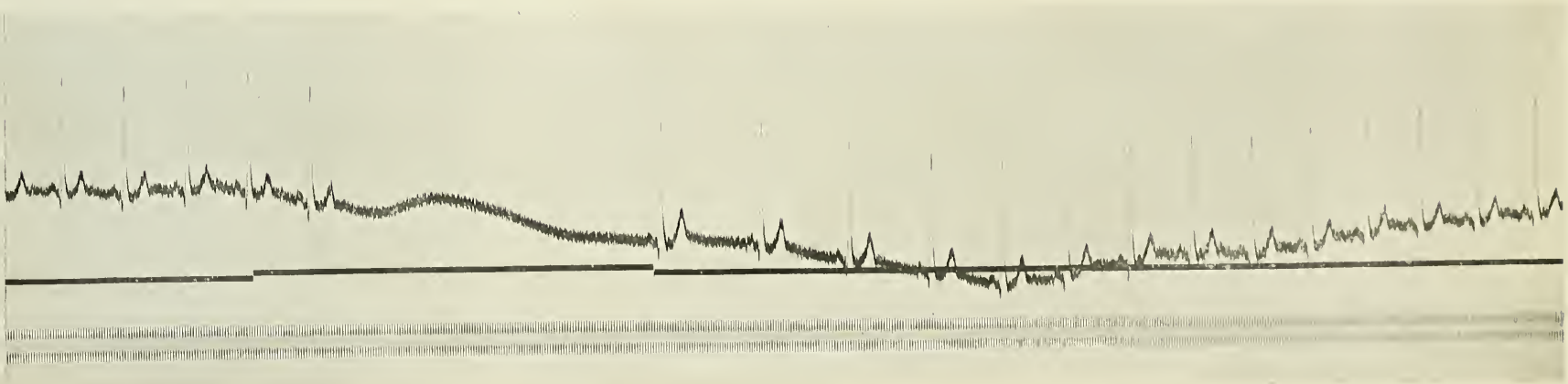


Fig. 85.—Compression of the right vagus during the period shown by the signal causes standstill of the whole heart for 4·4 seconds. Derivation II. 1 cm. = 1 millivolt. (*Quart. Journ. Med.*, vol. vi.)

1. Effects on the Auricles

I. *Rate.*—(1) *Compression of the vagus* may lengthen auricular diastole and thus retard the rate of the whole heart. The degree of auricular retardation thus induced varies considerably in different individuals, but the right vagus is almost invariably more effective than the left. A pronounced retardation of the whole heart by means of compression of the right vagus in shown in Fig. 84, obtained by derivation II. from a man aged 40. Before vagus compression, the duration of auricular diastole was 0·80-0·84 second, whereas the longer of the two auricular diastoles during the compression period lasted 2·31 second. A still more pronounced inhibition of the whole heart is shown in Fig.

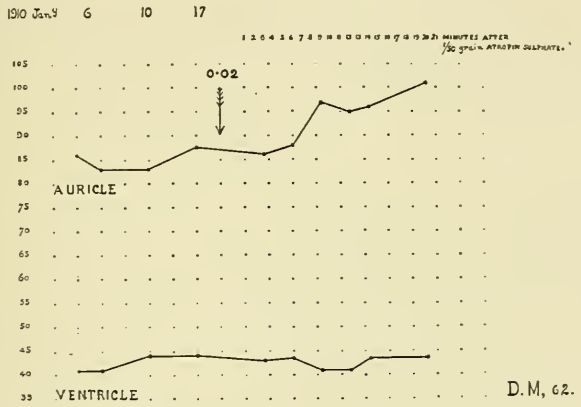


Fig. 86.—Diagram showing auricular acceleration after 0·02 grain of atropin sulphate. The ventricular rate is not increased. From a case of complete heart-block. (*Quart. Journ. Med.*, vol. vi.)

85, where compression of the right vagus caused standstill of the heart for 4·44 seconds. Similar effects in the human heart have been recorded by Wenckebach,[1] Laslett,[2] and others, and in the dog's heart by Einthoven,[3] and Rothberger and Winterberg.[4]

(2) *Atropin* paralyses the vagus endings in the heart, and thus accelerates the auricles. This effect is shown in Figs. 86 and 87. The

[1] Wenckebach, K. F., "Beiträge zur Kenntnis der menschlichen Herztätigkeit," *Arch. f. Anat. u. Physiol.* (Physiol. Abt.), Leipz., 1908, Suppl., 53.

[2] Laslett, E. E., "Syncopal Attacks, associated with Prolonged Arrest of the Whole Heart," *Quart. Journ. of Med.*, Oxford, 1908-9, ii., 347.

[3] Einthoven, W., "Le télécardiogramme," *Arch. internat. de Physiol.*, Liège, 1906-1907, iv., 132.

[4] Rothberger, C. J., and Winterberg, H., "Ueber die Beziehungen der Herznerven zur automatischen Reizerzeugung und zum plötzlichen Herztode," *Arch. f. d. ges. Physiol.*, Bonn, 1911, cxli., 343.

curves in Fig. 86 were constructed from a continuous record taken from a man, aged 62, who presented complete heart-block. The auricular rate was 87·5 per minute before the drug was given. Twenty minutes after the subcutaneous injection of 0·02 grain of atropin sulphate the auricular rate had risen to 101 per minute, but the ventricular rate did not become accelerated. A pronounced initial retardation, followed by acceleration, is shown in Fig. 87 from a man, aged 45, presenting digitalis heart-block. Before the test was performed the auricular rate varied from 83-85 per minute. The subcutaneous injection of 0·03 grain of atropin sulphate caused an initial retardation of auricular rate to 75 per minute. This passed off at the end of seven minutes, and was followed

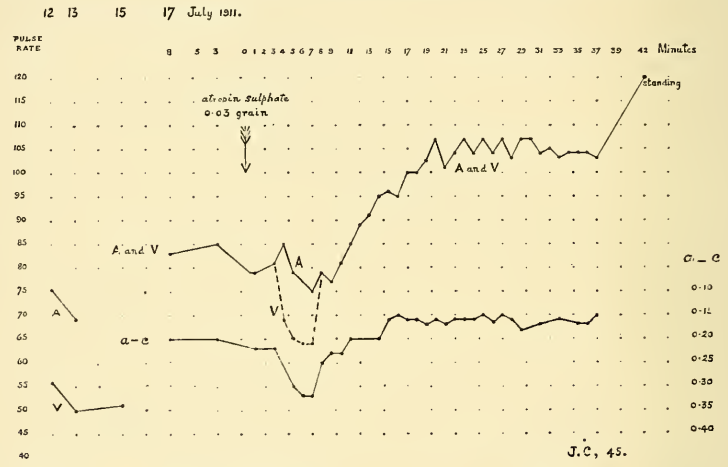


Fig. 87.—Diagram to show the rate per minute of auricles, *A*, and of ventricles, *V*, and the *a-c* interval before and after 0·03 grain of atropin sulphate in a case of digitalis heart-block. (*Quart. Journ. Med.*, vol. vi.)

by a progressive acceleration attaining a maximum of 107 per minute twenty minutes after injection of the drug. From the twenty-first until the thirty-seventh minute an auricular rate of 101-107 was maintained, rising to 120 when the patient rose up from the couch at the forty-second minute.

II. *Excitability.*—(1) *Compression of Vagus.*—Physiologists have demonstrated that excitation of the vagus in animals depresses the excitability of the auricular muscle (primary negative bathmotropic effect). Gaskell[1] states that this effect "is now universally allowed." It is difficult to estimate the excitability of the auricular muscle in man, because the methods applied by the physiologist to determine the thres-

[1] Gaskell, W. H., "The Contraction of Cardiac Muscle," *Schäfer's Text-book of Physiology*, Edin. and Lond., 1900, ii., 204.

hold of excitability are not applicable to the human heart. The only criterion which permits the clinician to assume that the excitability of the human auricle is increased is the occurrence of supra-ventricular extrasystoles, and even these indicate the presence of one or more unduly irritable foci in the auricular wall rather than of a general increase of excitability throughout the auricular muscle. An increased rate of auricular contraction is still less reliable evidence of heightened auricular excitability, because acceleration of rate may be due to other causes; for example, quicker rate of stimulus production, or shortening of the refractory phase.

In my own observations on the human heart I have never observed any indication of auricular excitability being increased or depressed during compression of either vagus.

(2) *Atropin.*—As vagus stimulation in animals depresses auricular excitability, it might be expected that atropin, by cutting off the vagus influences, would increase excitability. The drug might therefore be expected to promote the onset of supra-ventricular extrasystoles, but I have never observed this effect in man.

III. *Contractility.*—Physiologists are agreed that depression of auricular contractility is an early and striking effect of vagus stimulation and that the onset of, and recovery from, depression of contractility are usually gradual. Although the height of the summit *P* is not necessarily proportionate to the strength of auricular systole, we know that this deflexion is increased when the auricles are hypertrophied, or when both vagi have been cut. Einthoven[1] has demonstrated that in the dog during vagus stimulation the deflexion *P* may be diminished in size and become diphasic. Similar observations have been recorded in the dog's heart by Kahn,[2] Rothberger and Winterberg,[3] and others. My own observations on the human heart demonstrate the same effects. One record, from a man aged 55, suffering from mitral stenosis and presenting a loud, rough presystolic murmur, showed that compression of the left vagus, although retarding the rate of the heart very slightly, caused a notable reduction in the amplitude of the auricular deflexions. This may probably be regarded as indicating depression of auricular contractility. In Fig. 84 right vagus compression reduced the amplitude

[1] Einthoven, W., "Weiteres über das Elektrokardiogramm," *Arch. f. d. ges. Physiol.*, Bonn, 1908, cxxii., 517.

[2] Kahn, R. H., "Elektrokardiogrammstudien," *ibid.*, 1911, cxl., 627.

[3] Rothberger, C. J., and Winterberg, H., "Ueber die experimentelle Erzeugung extrasystolischer ventrikulärer Tachykardie durch Acceleransreizung," *ibid.*, 1911, cxlii., 461.

of the auricular deflexions and retarded the whole heart. In Fig. 88 the auricular deflexions *P*3 and *P*4 became of abnormal form. In view of Lewis's[1] experimental researches on the form of the auricular deflexion associated with primary negativity in different parts of the auricle, the inhibitory effect in Fig. 88 was probably most pronounced at the sinus node, and during the period of vagus compression some other part of the auricle initiated the stimulus for contraction.

(2) *Atropin.*—After section of both vagi in dogs, the height of the deflexion *P*, as demonstrated by Einthoven,[2] is increased nearly threefold. It might therefore be expected that atropin would increase the contractility of the human auricles, but I have not observed this effect. Their contractility may possibly be increased and yet the change be masked owing to the acceleration of rate.

IV. *Conductivity.*—That vagus stimulation may depress conductivity in the auricular walls was shown experimentally by Gaskell[3] and MacWilliam.[4] This change in the human heart, when slight, probably results in undue prolongation of auricular systole, and, when pronounced, in auricular fibrillation. Einthoven[2] has recorded the former effect in the dog, and Fig. 88 shows it in the human heart. In this figure the deflexions *P*3 and *P*4 last 0·245 and 0·280 second respectively, as compared with 0·105 second in the case of *P*8 after withdrawal of vagus compression.

We know that vagus stimulation depresses auricular conductivity and promotes the onset of auricular fibrillation. Winterberg[5] demonstrated this latter effect clearly, and showed that in a heart under the influence of atropin fibrillation ceases coincidently with the cessation of faradic stimulation, whereas in a heart without atropin fibrillation persists as an after-effect. It is therefore suggested that depression of auricular conductivity is an important factor in the production of auricular fibrillation. Digitalis and strophanthus have the same effect

[1] Lewis, T., "Galvanometric Curves yielded by Cardiac Beats Generated in Various Areas of the Auricular Musculature. The Pacemaker of the Heart," *Heart*, Lond., 1910-11, ii., 23.

[2] Einthoven, W., "Weiteres über das Elektrokardiogramm," *Arch. f. d. ges. Physiol.*, Bonn, 1908, cxxii., 517.

[3] Gaskell, W. H., "On the Innervation of the Heart, with Especial Reference to the Heart of the Tortoise," *Journ. of Physiol.*, Camb., 1883, iv., 43.

[4] MacWilliam, J. A., "On the Phenomena of Inhibition in the Mammalian Heart," *ibid.*, 1888, ix., 345.

[5] Winterberg, H., "Ueber Herzflimmern und seine Beeinflussung durch Kampher," *Zeitschr. f. exp. Pathol. u. Therap.*, Berlin, 1906, iii., 182; "Studien über Herzflimmern," *Arch. f. de ges. Physiol.*, Bonn, 1907, cxvii., 223; 1908, cxxii., 361.

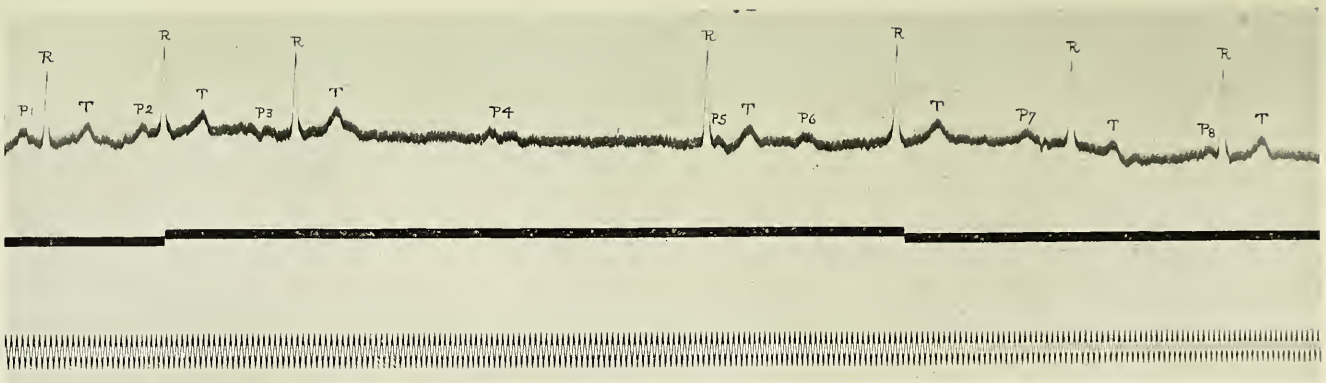


FIG. 88.—Compression of the right vagus during the period shown by the signal inhibits the whole heart and causes heart-block. Derivation II. 1 cm. = 1 millivolt.
(*Quart. Journ. Med.*, vol. vi.)

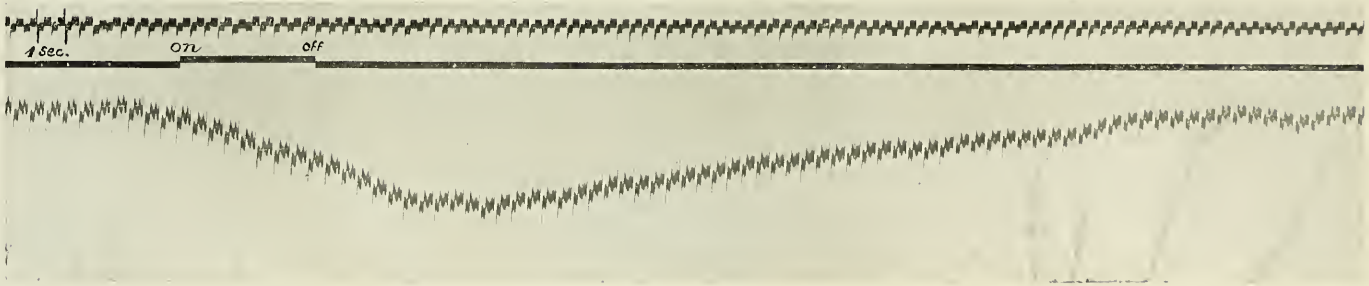


FIG. 89.—Pressure upon the right vagus, as shown by the signal, is accompanied and followed by a slow difference of potential. Derivation II. 1 cm. = 1 millivolt.
(*Quart. Journ. Med.*, vol. vi.)

as vagus stimulation in promoting the onset of auricular fibrillation, for, as will be shown later, these drugs tend to transform auricular flutter into fibrillation. From the preceding argument it is evident that they do so, probably by blocking the transmission of stimuli in the auricular walls.

V. *Tonicity.*—From Gaskell's[1] classical experiments we know that the tone of the heart muscle in the tortoise, frog, and toad is lowered by stimulation of the vagus and raised by stimulation of the augmentor nerve. Meek and Eyster[2] demonstrated a similar slow positive variation of potential in the heart of the tortoise under vagus stimulation, and some of my own records from the human subject show it likewise (Fig. 89). The change probably indicates depression of tone in the heart muscle under vagus stimulation.

2. Effects on the Atrio-ventricular Conducting System

From the work of Gaskell,[1] MacWilliam,[3] Muskens[4] and other physiologists it is well known that in the animal heart the conduction of stimuli to the ventricles may be impaired by means of vagus stimulation. When the human heart is beating with a physiological rhythm vagus compression seldom depresses the conductivity of the atrio-ventricular bundle unless there be some antecedent impairment of its function. An exceptional instance of vagus compression causing partial block in a healthy heart is shown in Fig. 88. In many cases of auricular flutter, however, compression of one or other vagus induces well-marked blocking of stimuli to the ventricles (see p. 104).

As yet we have little reliable information regarding the influence of vagus stimulation and paralysis on the excitability of the branches of the bundle. If those extrasystoles that are termed "ventricular" arise in the branches of the bundle, as seems probable, the onset or abolition of such extrasystoles by means of vagus stimulation or paralysis might yield information of interest and value. But our knowledge of this matter is still imperfect. One fact that we do know is that in cases of heart failure extrasystoles may disappear under the

[1] Gaskell, W. H., "On the Innervation of the Heart, with Especial Reference to the Heart of the Tortoise," *Journ. of Physiol.*, Camb., 1883, iv., 43.

[2] Meek, W. J., and Eyster, J. A. E., "Electrical Changes in the Heart during Vagus Stimulation," *Amer. Journ. of Physiol.*, Boston, 1912, xxx., 271.

[3] MacWilliam, J. A., "On the Phenomena of Inhibition in the Mammalian Heart," *ibid.*, 1888, ix., 345.

[4] Muskens, L. J. J., "An Analysis of the Action of the Vagus Nerve on the Heart," *Amer. Journ. of Physiol.*, Boston, 1898, i., 486.

influence of digitalis, one action of which is to stimulate the vagus. This restoration of a wholly normal rhythm, however, may be due to other factors than mere depression of excitability in the branches of the atrio-ventricular bundle. Moreover, the frequency of ventricular extrasystoles is seldom or never lessened by compression of the vagus, and in some instances their frequency is apparently lessened while the heart is under the influence of atropin.

3. Effects on the Ventricles

The vagi have but little direct influence on the ventricles. This is proved by the fact that in cases of complete heart-block the rate, rhythm, and strength of the ventricular beats are uninfluenced either by vagus stimulation or paralysis.

When the ventricles are responding in a physiological manner to each

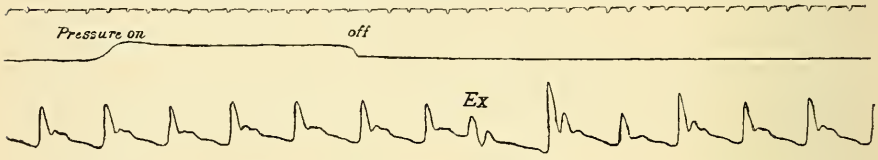


Fig. 90.—After compression of the right vagus there is one extrasystole (*Ex*), which is followed by transient alternation of the pulse.

supra-ventricular stimulus, however, the vagus acts upon the ventricles indirectly through the auricles, and the former seldom fail to follow the rate and rhythm set by the latter. In such instances vagus stimulation may occasionally depress the contractility of the ventricles, for the initial ventricular beats, after pronounced vagus inhibition, may only gradually attain their full strength. Under these circumstances a pulse tracing presents an ascending staircase.

4. After-effects of Vagus Stimulation

The inhibitory effects on the auricles usually pass off gradually, successive beats becoming quicker and stronger. In the atrio-ventricular conducting system the rate of recovery may be either slower or faster than in the auricles. The escape of the ventricles from vagus inhibition may be synchronous with that of the auricles, but in many instances the auricles are the first to escape.

The only other after-effect to which reference need be made is the

occasional onset of extrasystoles during the period of escape from vagus inhibition. They are probably due to sympathetic stimulation—for this is known to promote their onset. Moreover, the vagus contains sympathetic fibres, and the latent period of sympathetic stimulation is longer than that of inhibition (Fig. 90).

II. The Action of the Sympathetic on the Normal Heart

The sympathetic influences the heart in a manner the reverse of the vagus. The most striking effects of sympathetic stimulation are acceleration of rate, shortening of systole, and stronger contraction of auricles and ventricles. The conduction of stimuli to the ventricles is also improved. In electrocardiograms the height of the deflexions P and T is increased, whereas that of R is reduced. The quickened and forcible action of the heart under the influence of emotional disturbance and of substances such as adrenalin, and the action of the heart in exophthalmic goitre, are examples of sympathetic stimulation.

In other instances the physiological rhythm of the heart may be disturbed, for when the left accelerator is stimulated, according to Rothberger and Winterberg,[1] the left auricle may begin to contract before the right. This change is expressed by inversion of the auricular deflexion P. The same observers have shown that extrasystoles are liable to occur when the vagus and sympathetic are stimulated simultaneously.

III. The Action of the Vagus in Auricular Flutter

1. *The Action on the Auricles.*—It has already been mentioned that physiological beats of the auricles are under vagus control. They are retarded and weakened by vagus stimulation. In contrast thereto, it is found that in all cases of flutter in which compression of either vagus was recorded this failed to influence the auricles. Their rate and the form of their electrocardiographic deflexions remained unchanged. It has therefore been suggested by Cohn [2] that flutter may be due either to the inhibitory influences of the vagus failing to reach the sinus node, or to failure on the part of the node to respond to the vagus, and that

[1] Rothberger, J., and Winterberg, H., "Ueber die Beziehungen der Herznerven zur Form des Elektrokardiogramms," *Arch. f. d. ges. Physiol.*, Bonn., 1910, cxxxv., 506.

[2] Cohn, A. E., "Auricular Tachycardia, with a Consideration of Certain Differences between the Two Vagi," *Journ. of Exper. Med.*, New York, 1912, xv., 49.

in either case the dominant site of stimulus production is elsewhere than in the sinus node. The latter hypothesis is in harmony with MacWilliam's classical experiments on the production of flutter by faradic stimulation of the auricular appendix, and with the abnormal form of the auricular deflexion *P*, in nearly every recorded case of auricular flutter.

Digital compression of the vagus is a mild form of vagus stimulation. If stronger stimulation be applied, as in experimental work when the exposed vagus is faradised, this does not retard the flutter or cause the auricular beats to revert directly to a physiological rhythm, but induces either auricular fibrillation or a form of auricular action in which flutter and fibrillation are combined as described on p. 26. According to Robinson,[1] the normal rhythm may sometimes be restored several seconds after the cessation of vagus stimulation.

2. *The Action on the Atrio-ventricular Conducting System and the Ventricles.*—In most cases of auricular flutter in which the effects of vagus compression were recorded the ventricular rate was retarded although the auricles continued in flutter. This effect was not observed in Case I., but here there was complete heart-block. In Cases IV., VI., and L., likewise, compression of the vagus did not retard the ventricles, but the effect was seen in Cases II., III., V., XV., XVI., XVII., XXIV., XXV., and XXVI. The ventricular slowing is illustrated in Plates XIII. and XVIII. In both instances the escape from vagus inhibition is gradual.

In attempting to ascertain how the ventricular retardation is induced, we have to remember that it is similar to that obtained by MacWilliam[2] upon stimulation of the inhibitory area of the mammalian auricles (see p. 25). The retardation is not due to any lessening of the rate of the auricles nor, so far as can be determined, to any weakening of their beats. Moreover, it is almost certainly not due to any depression of ventricular excitability, for we know that vagus stimulation has little, if any, influence on this function of the ventricular muscle. Again, the effect cannot be ascribed to depression of contractility in the ventricles, for there is no ascending staircase during the period of escape from vagus inhibition; on the contrary, the arterial pulse waves are of large amplitude (Plate XIII.). We are therefore driven to

[1] Robinson, G. Canby, "The Influence of the Vagus Nerves on the Faradized Auricles in the Dog's Heart," *Journ. of Exper. Med.*, New York, 1913, xvii., 429.

[2] MacWilliam, J. A., "On the Phenomena of Inhibition in the Mammalian Heart," *Journ. of Physiol.*, Camb., 1888, ix., 345.

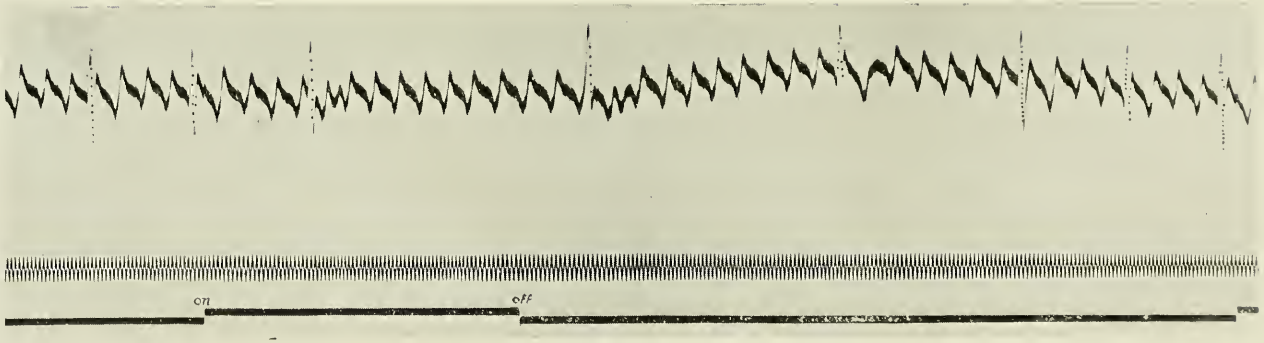


Fig. 91.—Auricular flutter at a rate of 274·2 per minute. Compression of the right vagus during the period shown by the signal retards the ventricles but not the auricles. Case II. Derivation II. 1 cm. = 1 millivolt.

the conclusion that the ventricular retardation obtained by compression of either vagus in cases of auricular flutter is due to depression of conductivity in the atrio-ventricular bundle whereby fewer stimuli—for example, 1 in 10 instead of 1 in 2 or 1 in 4—are transmitted to the ventricles.

IV. The Action of the Sympathetic in Auricular Flutter

Regarding this matter our knowledge is still imperfect. In some cases paroxysmal attacks of flutter are apparently brought on by emotional disturbance, excitement, or worry. In Case XXIII., for example, the patient, a clergyman, attributed many of his attacks to excitement, and found that they often followed his Sunday evening's sermon. But although in some cases sympathetic stimulation has a definite etiological relation to the onset of flutter, the sympathetic seems to have little, if any, influence on the auricles after flutter has been established. In each individual case the rate of the fluttering auricles usually remains remarkably constant, and is very slightly, if at all, affected by any form of psychical stimulation.

CHAPTER VIII

THE ACTION OF DRUGS OF THE DIGITALIS GROUP

I. Digitalis

Digitalis has been administered in about 50 per cent. of the recorded cases. When the auricles are in flutter the drug exerts a profound influence on the heart, and the effects are similar to those of vagus stimulation.

At first the drug does not check the flutter, but lowers the rate of the ventricles and often renders their rhythm irregular. The ventricles, which had previously been responding to every alternate auricular beat, may now respond to every third auricular beat so that the ventricular rate falls, for example, from 150 to 100 per minute, and the rhythm remains regular. In other instances the first recorded change is that the ventricles respond to a varying number of auricular beats. The rhythm of the ventricles then becomes irregular and their rate is somewhat lessened. This phase may be of short duration and is therefore liable to pass unnoticed or, because of the ventricular irregularity, to be mistaken for auricular fibrillation.

During the second phase the ventricles respond rhythmically to every fourth auricular beat, and consequently the ventricular rate falls to about 70-80 per minute. As a rule the patient now obtains much relief from all his distressing symptoms.

The third phase begins when the rapid rhythmic co-ordinate beats of the auricles become replaced by inco-ordinate fibrillar contractions. The rate of the ventricles is not necessarily much accelerated, and is indeed often under 80 per minute, but their rhythm becomes wholly irregular. In some instances the ventricular beats are coupled, and the pause after the second beat of each couple is then inconstant. In electrocardiograms the first beat of each couple is shown to be a response to a supra-ventricular stimulus, whereas the second beat has the character of a ventricular extrasystole. The second beat of each couple may fail to open the aortic valve, and the rate of the arterial pulse is then one-half of the true ventricular rate.

When the auricles pass into fibrillation no new symptoms arise.

On the contrary, the improvement in the patient's condition is usually maintained, and he may even feel remarkably well and be able to walk about without discomfort.

The fourth phase is characterised by restoration of the physiological rhythm. In some instances this may occur while the patient is still taking digitalis, but as a rule not until the drug has been discontinued. The restoration of normal rhythm has been observed as early as the third or as late as the twenty-third day after the patient has ceased taking digitalis. How long the physiological rhythm may be maintained we do not know, but in Case XIX. it was apparently maintained for at least three years. In other instances, however, the physiological rhythm is not maintained permanently, but is interrupted from time to time by paroxysms of flutter (Cases XVIII., XXIII.), flutter and fibrillation (Cases VI., XXXIV.), or occasional supra-ventricular extrasystoles (Case XXXIII.). Extrasystoles have also been recorded by Korteweg[1] as an after-effect of experimental auricular flutter in the cat.

All four phases are not constantly observed in every case of auricular flutter treated with digitalis. The first phase may be brief and pass unnoticed. The second phase is seldom omitted. The third phase—that of auricular fibrillation—may not develop for several reasons. The digitalis may not have been given in sufficient doses. In Case II. the patient took 1455 minims in the course of thirty-one days before the auricles passed into fibrillation. In Case XXIII. 485 minims were given in the course of nine days before fibrillation ensued. In other instances (Case XXIV., for example), the onset of nausea, vomiting, and diarrhœa may necessitate the drug being withdrawn before fibrillation is induced. Yet even in such cases the patient's symptoms may be relieved by the fall in the ventricular rate. Lastly, the physiological rhythm may never be restored fully, either because auricular fibrillation persists in spite of digitalis having been withdrawn, or because the degree of heart failure is so profound that the administration of the drug has to be continued.

II. Strophanthus

The action of strophanthus is essentially the same as that of digitalis. In Case III., with auricular flutter at a rate of 320 per minute, an intravenous injection of strophanthin induced auricular

[1] Korteweg, A. J., *Arhythmie door Atrium-fibrillatie*, Proefschrift, Leiden, 1913.

fibrillation, and lowered the ventricular rate from 160 to 96-104 per minute. On the following day the heart regained its physiological rhythm. In Cases XXIV. and XXV. strophanthus produced the same effects as had previously been obtained with digitalis.

III. Squill

Under the administration of squill, as recorded by Hay,[1] a ratio of As : Vs : : 3 : 1 changed to 4 : 1. Three weeks later the auricles were in fibrillation, and subsequently the normal rhythm was restored. In Case I. the auricular flutter was not converted into fibrillation by digitalis, strophanthus, or squill.

In auricular flutter the results obtained by the drugs of the digitalis group are similar to those of vagus stimulation. The initial effect of digitalis—retardation of ventricular rate alone—is identical with that of vagus compression. The subsequent conversion of auricular flutter into fibrillation, when the administration of digitalis is continued, is similar to the effect obtained experimentally on the fluttering auricles by faradisation of the vagus. There is consequently good reason for concluding that in auricular flutter digitalis acts by vagus stimulation, and that the primary effect of the drug is to depress the conductivity of the atrio-ventricular bundle, thus blocking the transmission of stimuli to the ventricles. This conclusion is strengthened by the fact that atropin may induce a transient abolition of the ventricular retardation, as recorded by Rihl (Case XV.), and Hume (Case XXX).

The essential similarity of action between vagus stimulation and digitalis leads us to the further conclusion that when the drug induces fibrillation, it does so by depressing conductivity within the auricular walls.

As we know that auricular flutter may be a phase of auricular activity intermediate between a physiological rhythm and fibrillation, the question arises whether there is a similar phase of flutter after fibrillation ceases and before the normal rhythm is restored, or whether the auricles revert directly from fibrillation to a normal rhythm. Certain clinical and experimental observations lend support to the latter hypothesis. According to Korteweg,[2] when fibrillation following experimental "auricular tachycardia" ceases, the action reverts to a slow, weak

[1] Hay, J., "Two Cases of Auricular Flutter," *Lancet*, Lond., 1913, ii., 986.

[2] Korteweg, A. J., *Arhythmie door Atrium-fibrillatie*, Proefschrift, Leiden, 1913.

co-ordinate contraction. Again, Lewis[1] points out that a rhythmic tachycardia induced by, and persisting after, stimulation of some portion of the heart may be abolished when a still faster rate of contraction, subsequently induced, has subsided. Moreover, it has been suggested by Cushny[2] that the period of slower contractions with pauses succeeding artificial acceleration of the isolated ventricles is due to depression of the function of stimulus production in the ventricular pacemaker, namely, to its fatigue. Similarly, when the physiological beats of the auricles have been replaced by fibrillation, and the latter in turn has ceased, a pause ensues before the physiological beats begin again. This pause must be regarded as indicating either fatigue, or inhibition of the physiological pacemaker by the fibrillation. When auricular fibrillation succeeds flutter, the sites wherein both the fluttering and the physiological beats were initiated might be supposed to suffer fatigue, and if the former site were the more fatigued the latter might be the first to recover its function after fibrillation ceases. Under such circumstances the normal rhythm of the heart would be restored.

Although in some instances there may be a direct change from fibrillation, following flutter, to a normal rhythm, this sequence has not yet

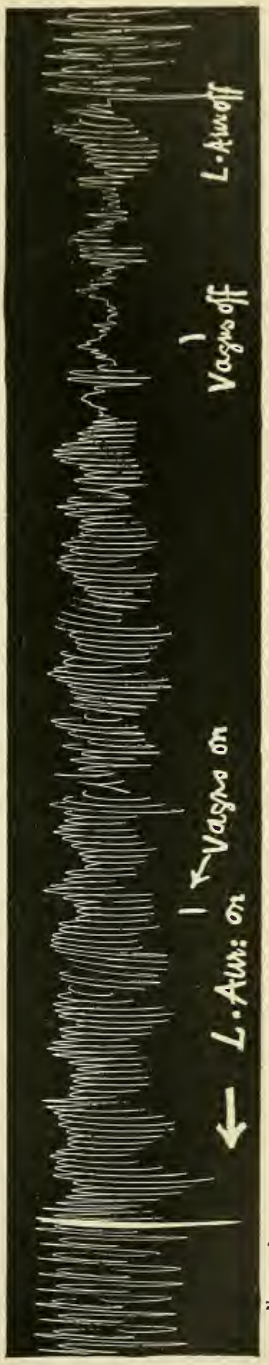


FIG. 92.—Cat's auricle. Faradisation of left auricle and stimulation of the left vagus. When the fibrillation succeeding flutter ceases there is again a period of flutter and then a brief pause before the normal rhythm is restored (W. E. Hume).

[1] Lewis, T., "Observations upon a Curious and not uncommon Form of Extreme Acceleration of the Auricle. 'Auricular Flutter,'" *Heart*, Lond., 1912-13, iv., 171.

[2] Cushny, A. R., "Stimulation of the Isolated Ventricle, with Special Reference to the Development of Spontaneous Rhythm," *Heart*, Lond., 1911-12, iii., 257.

been recorded clinically. It is not improbable that in some instances there may be an intermediate phase of flutter, so brief that it has uniformly escaped recognition in the human heart. In the cat's heart this brief phase has been recorded by W. E. Hume, who has kindly permitted me to make use of the record in Fig. 92. While the auricles were in flutter under the influence of weak faradic stimulation applied to the left auricle, the left vagus was faradised and fibrillation ensued. When the vagus stimulation was discontinued, the fibrillation became replaced by flutter; when the faradisation of the auricle ceased the flutter terminated, and after a short pause the normal rhythm was re-established.

CHAPTER IX

DIAGNOSIS

Whenever a patient complains of sudden attacks of palpitation and dyspnœa, with or without precordial pain, syncope, or other symptoms, and the pulse-rate during the attack attains or exceeds 130 or 140 per minute, auricular flutter should be suspected. If the pulse during the attack is absolutely rhythmic, the condition is probably due to flutter. Moreover, if the attack of rhythmic tachycardia comes on suddenly, and either ends abruptly or the rate of the rhythmic pulse-beats falls to one-half, while the patient is taking digitalis, the auricles are almost certainly in flutter. In many instances paroxysmal or persistent attacks of auricular flutter have probably been misinterpreted as auricular fibrillation, or have been designated by the vague term "paroxysmal tachycardia." Errors in diagnosis are especially liable to arise when the ventricular rhythm is irregular, because auricular fibrillation is then closely simulated. In such cases a correct diagnosis can seldom be made without the aid of the polygraph. Even this may fail to differentiate between flutter and fibrillation, and the true action of the auricles may not be revealed until electrocardiograms have been obtained.

The conditions for which auricular flutter is liable to be mistaken are: 1. Physiological rhythm without acceleration; 2, physiological rhythm with acceleration; 3, extrasystolic arrhythmia; 4, nodal rhythm; and 5, auricular fibrillation.

I. Physiological Rhythm without Acceleration

When the auricles are fluttering, and the ventricular rate is constantly one-third or one-fourth of the auricular rate, the cardiac impulse and the arterial pulse are rhythmic and their rate is not notably accelerated. In Case XXX., for example, the ventricular rate was usually about 87 per minute. In Case II., the rate was 68-72 for sixteen days while the patient was taking digitalis; and in Case XII., with partial heart-block, the ventricular rate was 42 when the auricular rate was 200 per minute. It is therefore evident that

auricular flutter may readily be overlooked when the patient is taking digitalis or when there is partial heart-block. Under these circumstances it may be impossible to determine, without the aid of instrumental records, whether the rhythm is physiological or whether the auricles are in flutter.

Two observations may be helpful. Firstly, the patient should be directed to breathe deeply. When the auricles are in flutter the rate of the ventricles does not hurry with inspiration and slow with expiration, as often occurs, in more or less marked degree, when the cardiac rhythm is physiological. Secondly, the patient should be asked to make some comparatively slight physical effort, such as is entailed by sitting up in bed or walking across the room. In a heart with a normal rhythm this effort, though usually causing some acceleration, does not cause sudden doubling of the ventricular rate, say from 80 to 160 per minute, whereas this instability and tendency to sudden doubling of the ventricular rate are not infrequent in auricular flutter.

Polygraph records are usually of much assistance in diagnosis. In a case of auricular flutter with a ratio of As : Vs :: 3 : 1, the series of three auricular waves for each beat of the arterial pulse may, however, be regarded erroneously as the *a*, *c*, and *v* waves of a physiological rhythm. This fallacy has been discussed by Hume,[1] who showed that in the case of flutter which he recorded the third wave was not a pure *v* wave because its summit was attained after the opening of the tricuspid valve. In this group of cases the first of the three waves is a pure *a* wave; the second is composed of *a* and *c*, and in contrast with a pure *c* wave has usually a more or less rounded summit; the third wave represents *a* and *v* waves fused in one.

Again, when the rate of the ventricles is constantly one-fourth of the auricular rate, a physiological rhythm may be simulated. In Case II., for example, the cardiac impulse and arterial pulse were rhythmic, at a rate of 68-72 for sixteen days, although the auricles were meanwhile in flutter at a rate of 268-276 per minute. The four waves *c*+*a*, *a*, *a*, and *a* in the jugular tracing (Fig. 78) might be mistaken for *c*, *v*, *h*, and *a* waves of a normal rhythm. In a normal rhythm, however, the summits of the four waves are not uniformly equidistant from one another. For example, in Fig. 7 the interval between *c* and *h* is constant, whereas that between *h* and *a*, or *h* and the succeeding *c*, is inconstant. Moreover, the rounded summit of the compound wave

[1] Hume, W. E., "A Case in which a High Speed of the Auricles did not produce Tachycardia," *Quart. Journ. of Med.*, Oxford, 1912-13, vi., 235.

$c+a$ in auricular flutter differs from the sharp-pointed summit of the wave c in a normal rhythm.

II. Physiological Rhythm with Acceleration (Sinus Tachycardia)

This is the most frequent form of tachycardia. It may be due to physiological causes such as physical exertion and emotion influencing a perfectly healthy heart, or to pathological causes. As examples of the latter, mention may be made of the various acute exanthemata, lobar pneumonia, pulmonary tuberculosis, exophthalmic goitre, acute endocarditis and myocarditis, and chronic valvular disease with cardiac dilatation and dropsy.

When the heart, in spite of acceleration, maintains its physiological rhythm, the onset and termination of the tachycardia are both gradual,

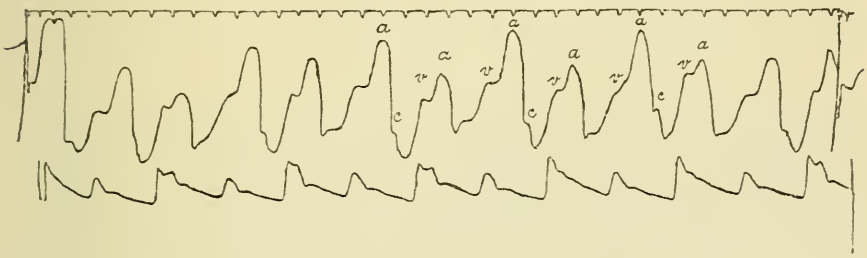


Fig. 93.—Sinus tachycardia, at the rate of 97 per minute, and pulsus alternans in a man, aged 57, with heart failure. Jugulo-carotid and brachial tracings. The a-c interval is 0·24 second.

and the rate seldom exceeds 140 per minute, even although there be marked myocardial enfeeblement. Thus in fifteen cases of exophthalmic goitre I found maximum rates of 83, 90, 97, 100, 107, 110, 114, 115, 117, 122, 128, 129, 131, 147 and 148 per minute. In none of these cases was the As—Vs interval less than 0·13 second, or more than 0·17 second; it was usually 0·14 second—the normal interval. Again, in sub-acute endocarditis with a physiological rhythm the rate seldom rises to 150 or 160 per minute, but I have seen cases in which the latter rate was attained. Moreover, I have seldom observed the ventricular rate to exceed 135 per minute in cases of chronic valvular disease so long as the physiological rhythm was maintained.

Polygraph records and electrocardiograms are of aid in diagnosis. When the heart's rhythm is physiological and the rate greatly accelerated, each carotid impulse in a jugular tracing is preceded by only one auricular wave. Owing to the curtailment of ventricular diastole each auricular wave is partially, or wholly, fused with the v wave of the

antecedent ventricular beat (Fig. 93). In an electrocardiogram there is likewise one deflexion P for each group of ventricular deflexions, and P is superposed on the antecedent T deflexion because of the shortened ventriculo-diastolic phase (Fig. 95). The As—Vs interval, as represented by the $P-Q$ interval, is seldom less than 0·14 second—the normal interval—even when the heart's rate is 120 or 130 per minute.

The superposition of a upon v, and of P upon T, implies that the auricles begin to contract before the antecedent ventricular systole has terminated. As the auricles contract at a time when the auriculo-ventricular valves are closed they cannot drive onwards the blood they contain. It therefore follows that any pre-existing mitral presystolic murmur disappears, and that each auricular systole probably causes a retrograde wave in the great veins, as shown by the large wave $v+a$ in

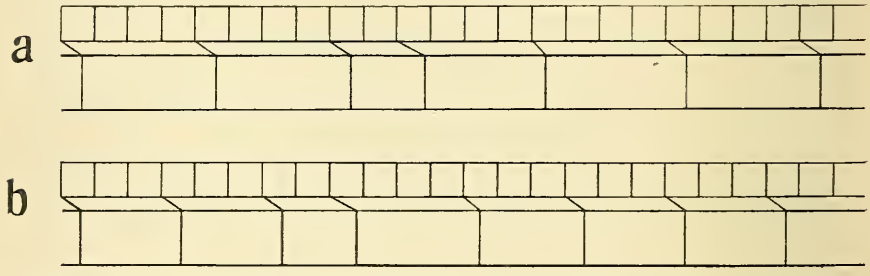


Fig. 94.—Diagrams to show that when the auricles are in rhythmic flutter irregularity of the ventricular responses may simulate (*a*) an auricular extrasystole with a pause that is not fully compensatory, or (*b*) a ventricular extrasystole with a fully compensatory pause.

the jugular tracings. It is thus evident that when the ventricular rate is greatly accelerated marked pulsation of the jugular veins and disappearance of a mitral presystolic murmur do not necessarily indicate loss of the physiological rhythm.

III. Extrasystolic Arrhythmia

Auricular flutter is most liable to be mistaken for extrasystolic arrhythmia when the patient is taking digitalis, yet before a ratio of As : Vs : : 4 : 1 is fully developed. A supra-ventricular extrasystole, with a pause that is not fully compensatory, is simulated when a constant ratio of 4 : 1 is momentarily interrupted by a ratio of 2 : 1, as in Fig. 94, *a*. In Case II. the auriculo-ventricular ratio was, at one time, frequently changing from 4 : 1 to 2 : 1, and the regular coupling of the cardiac impulse and arterial pulse used to suggest a regularly recurring supra-ventricular extrasystole (Fig. 97). A ventricular extrasystole

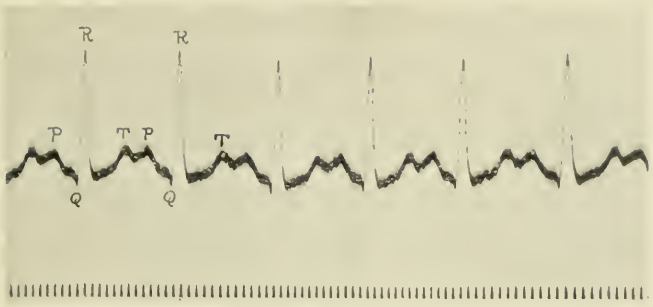


Fig. 95.—Sinus tachycardia at a rate of 131 per minute in a case of aortic and mitral incompetence. Derivation III. 1 cm. = 1 millivolt.

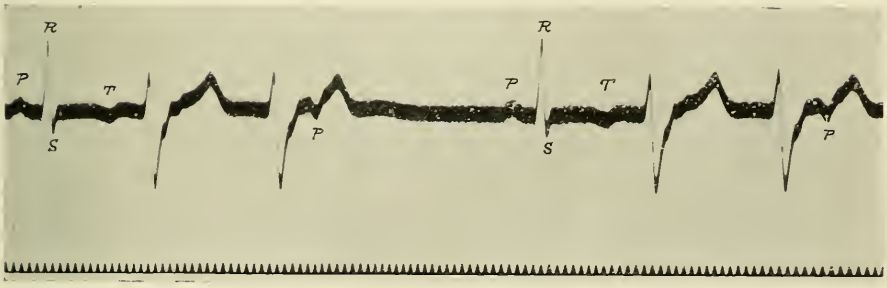


Fig. 96.—Each physiological beat is followed by two extrasystoles, in the second of which the auricular deflexion *P* is inverted. Male, aged 49. Derivation II.

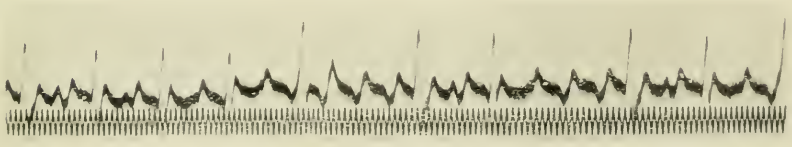


Fig. 97.—Auricular flutter at a rate of 281 per minute. The ventricles usually respond to every second auricular beat, but occasionally to every fourth. Case II. (18th August 1912). Derivation II. 1 cm. = 1 millivolt.

would be simulated if the auriculo-ventricular ratio changed momentarily from 3 : 1 to 2 : 1, 4 : 1, and then back to 3 : 1 (Fig. 94, *b*). In cases of this nature polygraph tracings or electrocardiograms will establish the diagnosis.

Auricular flutter may be simulated still more closely by a paroxysmal attack in which each beat is a ventricular extrasystole. This is a somewhat rare form of paroxysmal tachycardia. In Fig. 96, from a man aged 49, extrasystoles occur in pairs after each physiological beat. In the second beat of each pair the auricular deflexion is inverted, and there is a long post-extrasystolic pause before the next physiological beat. In another case, that of a man aged 39 who was suffering from aortic and mitral incompetence, the ventricular extrasystoles were sometimes "single," and at other times there were series of two, three, four, five, or more extrasystoles in rapid succession, only

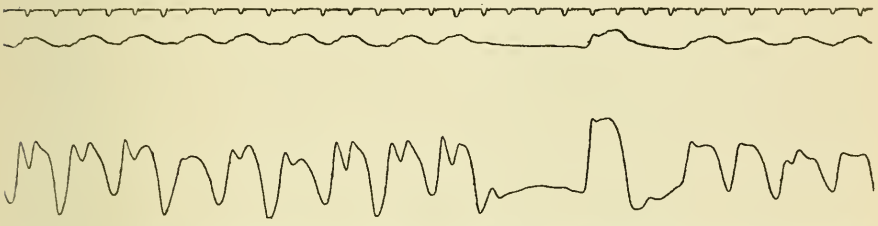


Fig. 98.—Paroxysmal tachycardia due to ventricular extrasystoles in a case of aortic and mitral incompetence. Brachial and apical tracings. The last nine beats of a long paroxysm are shown. After the post-extrasystolic pause, which is fully compensatory, there are two normal beats and thereafter another paroxysm begins.

the last beat of each series being followed by a post-extrasystolic pause. Fig. 98, from this case, shows the end of a long paroxysm of extrasystoles at a rate of 153 per minute. This paroxysm is succeeded by a fully compensatory post-extrasystolic pause. Two normal beats then occur, and thereafter another paroxysm of extrasystoles begins. In the case described by Stuart Hart [1] there were similar paroxysms, lasting from a few seconds to three minutes, in which ventricular extrasystoles recurred rhythmically at a rate of 240 per minute. Hoffmann [2] has also recorded a case of this nature.

IV. Nodal Rhythm

A nodal extrasystole, as defined on p. 15, is a premature and synchronous beat of the auricles and ventricles. A nodal rhythm is a series of rhythmic or arrhythmic beats in which the auricles and ventricles contract simultaneously. Each ventricular beat may start

[1] Hart, T. Stuart, "Paroxysmal Tachycardia," *Heart*, Lond., 1912-13, iv., 128.

[2] Hoffmann, A., *Die Elektrographie*, Wiesbaden, 1914, Case xix., 250-252.

before the auricles begin to contract; the auricles and ventricles may begin to contract at the same moment; or each auricular contraction may precede that of the ventricles, but in this case the As – Vs interval is much shorter than normal.

The site of origin of the dominant stimuli in nodal rhythm has been the subject of much discussion. It is not in the ventricles, because electrocardiograms demonstrate that these chambers are contracting in response to supra-ventricular stimuli. Moreover, the site of stimulus production is presumably not in the general auricular musculature, the sinus node, or the wall of the coronary sinus, because if the stimuli did arise in these situations it would have to pass through the atrio-ventricular node before reaching the ventricles, and it is in this node, according to Hering,[2] that the transmission of supra-ventricular stimuli suffers delay. The synchronism of auricles and ventricles in nodal extrasystoles and nodal rhythm suggests that the stimuli for contraction are generated at a site approximately midway between these chambers, and presumably in the atrio-ventricular node or bundle. According to this hypothesis nodal rhythm results from the acquirement by these structures of the power of initiating stimuli for contraction at a greater rate than the sinus node. The functional activity of the atrio-ventricular node or bundle has become unduly exalted, or that of the sinus node has become depressed, or both changes have occurred simultaneously.

Nodal rhythm was described by Lohmann [1] as resulting from stimulation of the septum in the region of the node and bundle; by Lohmann [3] and Hering,[4] as a result of destructive lesions at the cavo-auricular junction; by Rihl [5] and Hering [6] as an effect of accelerator stimulation; and by Cushny,[7] after injection of dogs with aconitine. The site of

[1] Lohmann, A., "Zur Automatie der Brückenfasern und der Ventrikel des Herzens," *Arch. f. Anat. u. Physiol.* (Physiol. Abt.), Leipz., 1904, 431.

[2] Hering, H. E., "Nachweis, dass die Verzögerung der Erregungsüberleitung zwischen Vorhof und Kammer des Säugethierherzens im Tawara'schen Knoten erfolgt," *Arch. f. d. ges. Physiol.*, Bonn, 1910, cxxxi., 572.

[3] Lohmann, A., "Ueber die Funktion der Brückenfasern an Stelle der grossen Venen die Führung der Herztätigkeit zu übernehmen," *ibid.*, 1908, cxxiii., 628.

[4] Hering, H. E., "Experimentelle Studien an Säugethieren über das Elektrokardiogramm," *ibid.*, 1909, cxxvii., 155.

[5] Rihl, J., "Experimentelle Analyse des Venenpulses bei den durch Extrasystolen verursachten Unregelmässigkeiten des Säugethierherzens," *Zeitschr. f. exp. Pathol u. Therap.*, Berlin, 1905, i., 43.

[6] Hering, H. E., "Einiges über die Ursprungsreize des Säugetierherzens und ihre Beziehung zum Accelerans," *Zentralb. f. Physiol.*, Leipz., 1905, xix., 129.

[7] Cushny, A. R., "The Irregularities of the Mammalian Heart observed under Aconitine and on Electrical Stimulation," *Heart*, London, 1909-10, i., 1.

origin of the stimuli has been discussed in a critical paper by Erlanger.[1] Among the most instructive experimental observations are those of Rothberger and Winterberg.[2] These observers found that in almost every instance the accelerator fibres passing to the cavo-auricular junction could be put out of action by the local application of ethyl chloride spray, and that subsequent stimulation of the left accelerator uniformly induced a nodal rhythm with synchronous contractions of the auricles and ventricles. While the nodal rhythm was developing, the auricular deflexions became gradually merged in those of the ventricles until eventually, when the auricles and ventricles were beating synchronously, the auricular deflexions were no longer visible. The jugular pulsations were then of strikingly large amplitude. This phenomenon may also be observed in cases of nodal rhythm in man (see Fig. 99).

The most notable cases of nodal rhythm in the human heart are those described by Cowan[3] and by Hoffmann.[4] In Cowan's cases the cardiac rhythm was regular, and the rate varied, being sometimes very frequent (over 200 per minute), but in other instances only slightly accelerated. The nodal rhythm was either intermittent and paroxysmal, or persistent. In all the cases recorded by Cowan the $a-c$ interval in the jugular tracings was notably shorter than normal. Cowan, Fleming, and Kennedy,[5] have described inflammatory changes in the atrio-ventricular node of six cases of acute endocarditis with persistent nodal rhythm. This rhythm has also been described by Rihl[6] and Lewis;[7] it has been observed in cases of rheumatism, typhoid fever, and scarlatina by Belski,[8] and in cases of diphtheria by Hecht[9] and

[1] Erlanger, J., "The Localization of Impulse Initiation and Conduction in the Heart," *Arch. of Intern. Med.*, Chicago, 1913, xi., 334.

[2] Rothberger, C. J., and Winterberg, H., "Ueber die Beziehungen der Herznerven zur atrio-ventrikulären Automatie (nodal rhythm)," *Arch. f. d. ges. Physiol.*, Bonn, 1910, cxxxv., 559.

[3] Cowan, J., *Diseases of the Heart*, Lond., 1914, 133-158.

[4] Hoffmann, A., "Die Elektrographie als Untersuchungsmethode des Herzens und ihre Ergebnisse," Wiesbaden, 1914, 202-247.

[5] Cowan, J., Fleming, G. B., and Kennedy, A. M., *Trans. XVIIth Internat. Congress of Med.*, London, 1914, Sect. VI. Med., Part II., 223.

[6] Rihl, J., "Ueber atrioventrikuläre Tachycardie beim Menschen," *Deutsch. med. Wochenschr.*, 1907, xxxiii., 632.

[7] Lewis, T., "Auricular Fibrillation and its Relationship to Clinical Irregularity of the Heart," *Heart*, Lond., 1909-10, i., 306.

[8] Belski, A., "Beobachtungen über atrioventrikuläre Automatie im Verlauf der Infektionskrankheiten," *Zeitschr. f. klin. Med.*, Berlin, 1909, lxvii., 515.

[9] Hecht, A. F., "Ueber atrioventrikuläre Automatie bei post-diphtherischer Herzschwäche," *Wien. med. Wochenschr.*, 1912, lxii., 2015.

Hume.[1] Nodal rhythm may be the precursor of, or may alternate with, either auricular flutter or fibrillation.

The Venous Pulse.—In clinical work the differentiation of auricular flutter from nodal rhythm is not yet possible without the aid of polygraph tracings or electrocardiograms. In the former, the apical impulse begins before or simultaneously with the auricular wave in the jugular

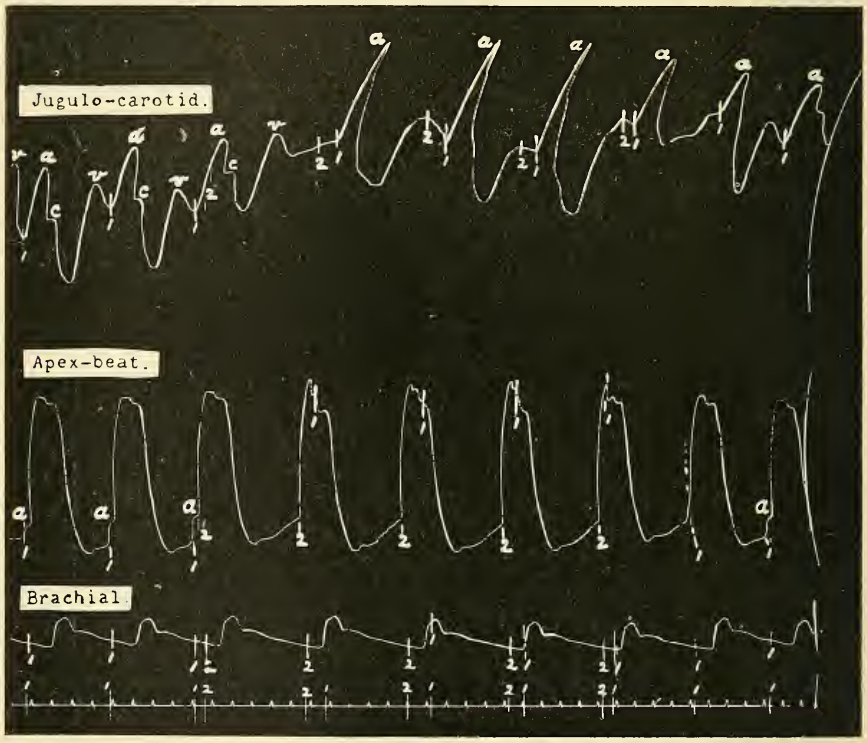


Fig. 99.—Nodal rhythm beginning abruptly and passing off gradually. In the first three beats and in the last beat the contraction of the auricles (1) precedes that of the ventricles (2); whereas in the nodal beats the ventricles begin to contract before the auricles, and the auricular wave, *a*, in the jugular vein is of large size. While the nodal rhythm is passing off, the interval 2-1 becomes progressively shorter until, eventually, the contraction of the auricles again precedes that of the ventricles.

tracings, or the interval between the contraction of auricle and ventricle is notably short. In consequence of the auricles being in contraction at the same time as the ventricles, the former cannot expel their blood into the latter, and the auricular wave in the jugular veins is therefore of large size. The onset of nodal rhythm is well shown in Fig. 99, obtained from a joiner aged 47, who was suffering from sub-acute

[1] Hume, W. E., "A Polygraphic Study of Four Cases of Diphtheria, with a Pathological Examination of Three Cases," *Heart*, London, 1913-14, v., 25.

myocarditis with cyanosis, dyspnœa, and other signs of heart failure. When the patient first came under observation the pulse-rate was usually about 70 per minute; the rhythm was normal and the venous tracing presented distinct *a*, *c*, and *v* waves, as in the first three beats of Fig. 99. In the next five beats of Fig. 99, however, the rhythm is not normal, because the ventricles begin to contract before the auricles. In the apical tracing the auricular wave is no longer visible, whereas in the venous tracing it is of large size. In the first and second beats of the nodal rhythm (the 4th and 5th beats recorded in Fig. 99), the Vs – As interval is 0·20 and 0·25 second respectively. Thereafter the Vs – As interval becomes gradually shorter with each successive beat, until eventually the auricular contraction again precedes that of the ventricles. This gradual transition from a nodal to a normal rhythm is similar to that recorded by Rothberger and Winterberg.[1]

For a month after the record in Fig. 99 was obtained the heart's rhythm varied from normal to nodal. With a normal rhythm the rate was about 90, with a nodal rhythm about 79 per minute. The patient's general condition improved greatly, and he resumed his work. About one month later, however, he again became cyanosed, very breathless and dropsical. The auricles were then found to be in fibrillation. Cheyne-Stokes breathing developed, the dropsy became progressively worse, and the patient gradually sank and died three and a half months after he first came under my observation.

At the post-mortem examination all the chambers of the heart were found to be dilated; the left ventricle was hypertrophied and presented abundant patches of fibrosis. The valves were all healthy. The lungs showed chronic venous congestion and hypostatic pneumonia. On microscopic examination, the walls of all the chambers of the heart revealed widespread fibrosis and pronounced cellular infiltration. In the auricular septum there was much engorgement of the small vessels, and many hæmorrhages. The atrio-ventricular node and bundle were markedly involved by the inflammatory infiltration, but the branches of the bundle were implicated to a lesser degree.

Electrocardiograms.—The ventricular deflexions in nodal rhythm are essentially of normal character, indicating supra-ventricular stimulation. The auricular deflexion is either (1) synchronous with, and masked by the ventricular deflexions, or (2) precedes the initial ventricular deflexion

[1] Rothberger, C. J., and Winterberg, H., "Ueber die Beziehungen der Herznerven zur atrio-ventrikulären Automatie (nodal rhythm)," *Arch. f. d. ges. Physiol.*, Bonn, 1910, cxxxv., 559.

by an interval that is decidedly shorter than normal, and is of atypical form.

The first variety of nodal rhythm is illustrated in Fig. 100, obtained from a woman, aged 49, affected with mitral incompetence. For two years she had been suffering from dyspnœa and palpitation, and she had been dropsical. While she was in hospital her auricles were usually in fibrillation. On one occasion, however, all the beats were probably responses to stimuli arising in the atrio-ventricular conducting system, and as in the cases described by Eppinger and Stoerk,[1] Mathewson[2] and others, the form of the ventricular deflexions indicates that the stimuli were reaching the ventricles through one or other branch of the bundle, but not through both branches simultaneously. The stimuli for the beats lettered *X* in Fig. 100 probably originated in the right and left branch of the bundle, but in neither instance did the stimulus pass back to the auricles. In each of the remaining beats, however, the stimulus passed not merely forwards to the ventricles but also backwards to the auricles, as shown by the deflexion *P*.

The second variety of nodal rhythm is represented in Fig. 101. The patient was a Crimean veteran, aged 72, under the care of Professor Russell in the Royal Infirmary. For about six months the patient had been suffering from headache, dyspnœa, dropsy, and other symptoms of heart failure. Soon after his admission to hospital, orthopnœa became very intense, and the legs became swollen up like bolsters. The apex-beat was in the normal situation, but was unduly forcible. No murmurs were audible. The arteries of the arms were thick, and the systolic pressure was 200 mm. Hg. The cardiac rhythm was almost constantly irregular owing to numerous auricular extrasystoles, each of which yielded an inverted *P* deflexion and a short $P-R$ interval (0·10 second), whereas the corresponding interval of the normal beats was 0·14 second.

On several occasions when the heart was grossly irregular, each beat was initiated by an auricular contraction yielding an abnormal, inverted deflexion of the same form as that of the isolated auricular extrasystoles (Fig. 101). On these occasions the irregularity of the pulse was so pronounced as to simulate auricular fibrillation very closely. Under the influence of rest, digitalis, and squill, the patient made a satisfactory

[1] Eppinger, H., and Stoerk, O., "Zur Klinik des Elektrokardiogramms," *Zeitschr. f. klin. Med.*, Berlin, 1910, lxxi., 157.

[2] Mathewson, G. D., "Lesions of the Branches of the Auriculo-ventricular Bundle," *Heart*, Lond., 1912-13, iv., 385.

PLATE XX.

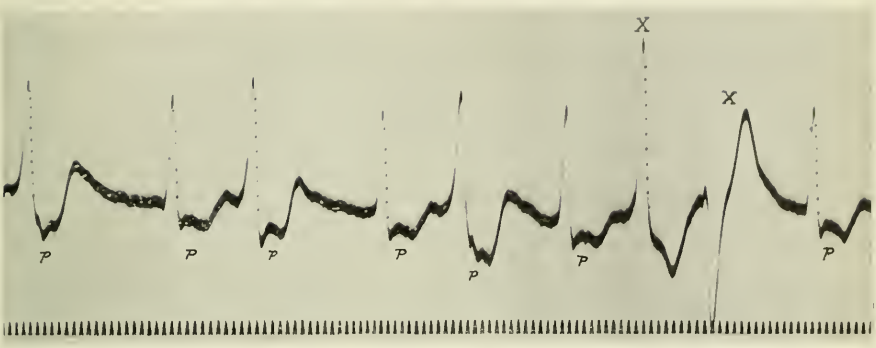


Fig. 100.—Nodal arrhythmia with two extrasystoles (*X*). From a woman, aged 49, with mitral incompetence. Derivation II. 1·5 cm. = 1 millivolt.

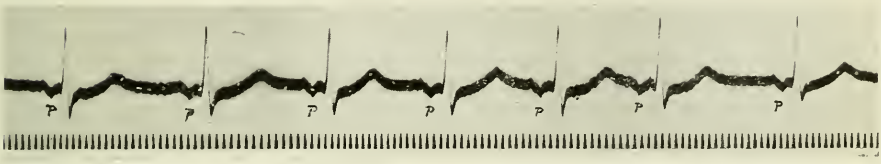


Fig. 101.—Nodal rhythm. The heart's action, at a rate of 95 per minute, is wholly irregular. The ventricles respond to auricular beats which are represented by the atypical deflections *P*. Derivation II. 1·5 cm. = 1 millivolt.

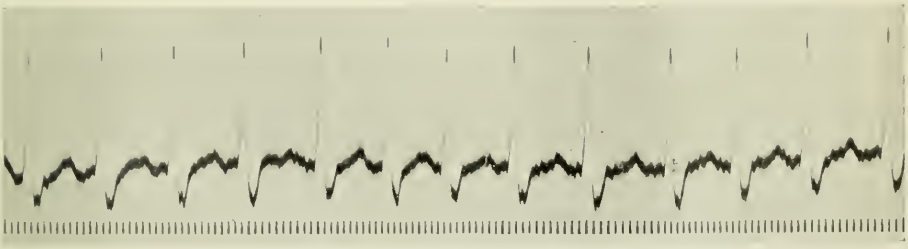


Fig. 102.—Auricular fibrillation with a ventricular rate of 160 per minute. Derivation II. 1 cm. = 1 millivolt.

recovery; he lost all his dyspnœa and dropsy; his heart regained its normal rhythm except for an occasional extrasystole; and in the course of a few months, when he was able to return home, he was in enjoyment of better health then that of many a man of his years.

In this case there was a heterotopic site of stimulus production giving rise, at first, to isolated supra-ventricular extrasystoles and subsequently to a series of beats similar in character and irregular in rhythm. The exact site of stimulation cannot be defined, for although, according to Lewis,[1] inverted auricular deflexions represent nodal stimulation this hypothesis is not established, and according to Rothberger and Winterberg[2] the auricular deflexion is of this form when the left, instead of the right, auricle initiates the contraction.

Rhythmic "paroxysmal tachycardia" is probably more often auricular flutter than nodal rhythm. In some of the cases recorded by Hoffmann[3] the sudden doubling, or halving, of the ventricular rate, and the ventricular retardation effected by compression of the vagus, suggest the possibility of auricular flutter. But the following was probably an example of paroxysmal nodal rhythm.

The patient was a woman, aged 33, without any history of rheumatism, who had suffered for eight years from occasional attacks of "flying of the heart." As a rule each attack was preceded by nausea and sickness, lasted for about one day, and passed off gradually. When she was admitted to the Royal Infirmary under the care of Dr. Chalmers Watson, "her heart had been flying for a week." During the first month of her residence in hospital a number of paroxysms were observed.

When the cardiac rhythm was normal, the rate was about 80-90 per minute, the arterial pulse was of good volume, and the jugular pulse was small. The heart was not enlarged and no murmurs were audible.

During the attacks, which lasted for a few hours to several days, the ventricles were beating rhythmically at a rate of 168-226 per minute; the pulse in the arteries of the arm became almost imperceptible whereas the pulsations of the jugular veins became so forcible as to resemble violent throbbing of the carotid arteries. The liver could

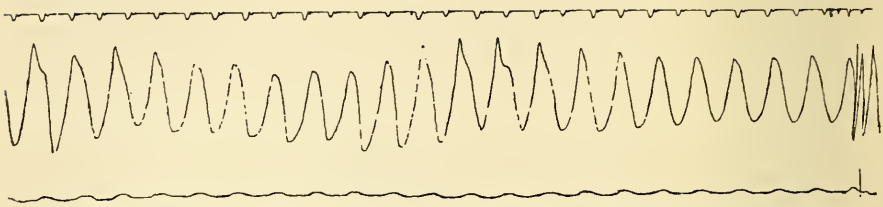
[1] Lewis, T., "Auricular Fibrillation and its Relationship to Clinical Irregularity of the Heart," *Heart*, Lond., 1909-10, i., 306.

[2] Rothberger, C. J., and Winterberg, H. "Ueber die Beziehungen der Herznerven zur atrio-ventrikulären Automatie (nodal rhythm)," *Arch. f. d. ges. Physiol.*, Bonn, 1910, cxxxv., 559.

[3] Hoffmann, A., "Neue Beobachtungen über Herzjagen," *Deutsch. Arch. f. klin. Med.*, Leipz., 1903, lxxviii., 39.

be felt to pulsate freely, the heart sounds acquired the fœtal rhythm, and if the attack was a prolonged one the patient became very breathless.

On two occasions an attack came on suddenly while the armlet of the polygraph was being adjusted. The ventricular rate rose abruptly from 90 to 195, and from 84 to 222, per minute. Fifteen minutes after



[Fig. 103.—Paroxysmal tachycardia with a rate of 224 per minute. Jugulo-carotid and brachial pulsations.

the second of these attacks had begun, the patient was questioned regarding her symptoms, and was found to be wholly unaware of the attack. On one occasion when the effect of change of posture was tested, the pulse-rate was found to rise from 168 to 226 per minute when the patient sat up in bed (Fig. 103). Compression of neither vagus slowed the pulse.

V. Auricular Fibrillation

Both in its paroxysmal and in its persistent forms, auricular flutter may be mistaken for fibrillation. As a rule the perfectly rhythmic action of the ventricles excludes the possibility of auricular fibrillation. But difficulty may arise when the ventricular rate is very fast or when the ventricular rhythm, in flutter, is irregular.

When auricular fibrillation is combined with a very rapid action of the ventricles — for example, 160-180 per minute — the irregularity is often so slight that it may be unrecognisable on palpation of the pulse and apex-beat or on auscultation of the heart-sounds.

Fig. 102 was obtained from a case of this nature. The patient was a tall, thin man, aged 61, who showed no trace of cyanosis or œdema. He had been short of breath for six weeks. The apex-beat was forcible but in normal position; the first sound at the apex was impure, the second sound was clear and accentuated. The arteries were moderately thickened, and the urine was albuminous. The ventricular rate was 159 per minute. Definite auricular deflexions, of small amplitude and irregular rhythm, characteristic of auricular fibrillation, were not visible except during an occasional long ventricular

diastole. As a rule the auricular deflexions were inconspicuous; nevertheless auricular fibrillation was indicated by the slight irregularity of the ventricles, and by the varying form of the curve between *S* and *R* in successive cycles. In cases of this nature, a little irregularity of the pulse suggesting auricular fibrillation can usually be recognised even by the finger alone.

Auricular fibrillation is simulated still more closely when the ventricles are responding irregularly to fluttering auricles; for example, when successive ventricular contractions are in response to 4, 3, and 2 auricular beats. This in itself renders the ventricular rhythm irregular, and the irregularity is intensified further by the fact the As–Vs interval is longer when the ratio of As : Vs is 3 : 1 than when it is 4 : 1 (Fig. 82). Under these circumstances, digital examination of the pulse, palpation of the apex-beat, and auscultation of the heart-sounds may fail to differentiate between flutter and fibrillation. In such cases compression of the right vagus may be helpful. In my experience this usually retards the ventricles when the auricles are in flutter, and frequently fails to do so when the auricles are in fibrillation. A pronounced ventricular retardation upon compression of one or other vagus consequently suggests flutter rather than fibrillation.

It sometimes happens that a greatly accelerated and markedly irregular action of the ventricles suddenly becomes absolutely rhythmic while the rate remains very fast, say 150 per minute. This change may occur frequently within the course of a few minutes. It indicates either that auricular fibrillation and flutter are alternating with each other, or that with persistent flutter the ventricular responses are sometimes irregular (for example, 3 : 1, 2 : 1, 4 : 1, 3 : 1) and sometimes regular (for example, 2 : 1, 2 : 1, 2 : 1).

Simple sphygmographic tracings may suffice to demonstrate that the arterial pulse-beats are wholly arrhythmic, indicating fibrillation, or that groups of beats are of identical duration, indicating a rhythmic auricular action (Figs. 75 and 104). In auricular fibrillation the height of the pulse-wave in a sphygmographic tracing bears no constant relation to the length of the preceding pulse period. This has been regarded as a definite indication of auricular fibrillation. The same inconstant relation, however, is often observed when the auricles are fluttering and the ventricles responding to a varying number of auricular beats (Fig. 104).

If polygraph tracings reveal a rhythmic series of large auricular waves in the jugular veins at times when the ventricles are in diastole,

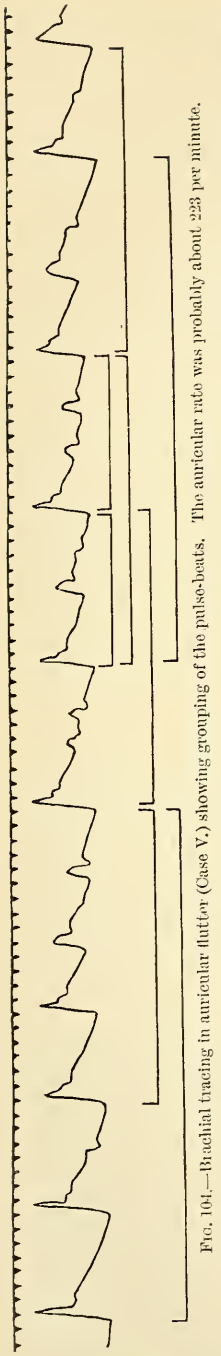


Fig. 104.—Brachial tracing in auricular flutter (Case V.) showing grouping of the pulse-beats. The auricular rate was probably about 223 per minute.

the condition is clearly flutter and not fibrillation. Even although the auricular waves in the jugular pulse are not large, the auricles are almost certainly in flutter if the arterial pulse-beats occur in groups of equal length, as in Fig. 104. On the other hand, if the auricular waves are small, irregular in rhythm and very rapid, and if at the same time there is little or no uniformity in the grouping of the arterial pulse-beats, the auricles are either in fibrillation or in the form of activity represented by simultaneous flutter and fibrillation. Again, if the jugular pulse is of the ventricular form and the rhythm of the ventricles is wholly irregular the auricles are in fibrillation, whereas a jugular pulse of the same form associated with a rapid and rhythmic action of the ventricles almost certainly indicates auricular flutter.

Electrocardiograms afford the most reliable means of diagnosis, but unfortunately they are not always available. In almost every recorded case of flutter the auricular deflexions are of constant form, rhythmic, large and comparatively infrequent at a rate of about 200-370 per minute. In fibrillation the auricular deflexions are irregular in form and rhythm, of small amplitude and greater rate (380-520 or more per minute). The distinctive features are usually seen best in records taken by derivation III. (left hand and left foot), and with a comparatively slack fibre. If the auricular deflexions are obscured as a result of excessive ventricular acceleration, their true form may often be revealed by means of compression of the right vagus in the neck (Plate XIII., Fig. 65).

The most difficult cases of all are those in which the auricular action is frequently changing from flutter to fibrillation, and those in which the auricular action is a combination of flutter and fibrillation as

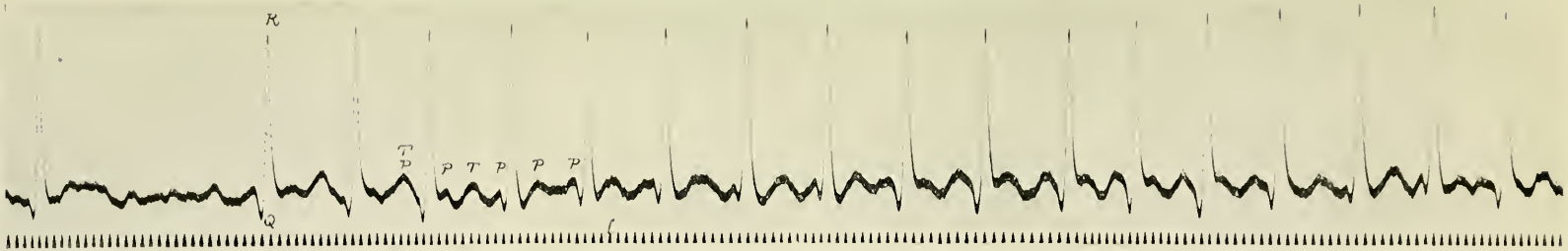


Fig. 105.—Auricular fibrillation passing into flutter at a rate of 377·2 per minute, with a ratio of As : Vs : : 2 : 1. Case V. Derivation II. 1·5 cm. = 1 millivolt. (*Quart. Journ. Med.*, vol. vii.)

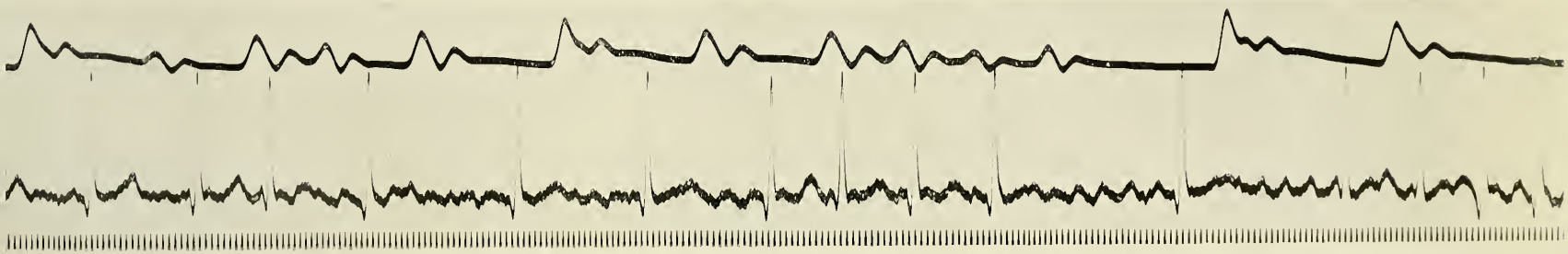


Fig. 106.—Auricular fibrillation becoming coarser and eventually passing into flutter. Case V., brachial pulsations and electrocardiogram by derivation III. 1 cm. = 1 millivolt. (*Quart. Journ. Med.*, vol. vii.)

described on pages 26, 93, and 94. The clinical features in the latter cases resemble auricular fibrillation more closely than flutter.

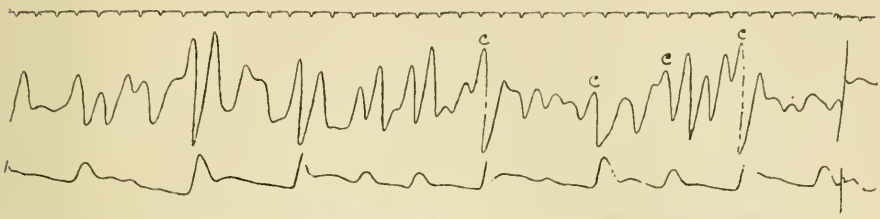


Fig. 107.—Auricular flutter with marked irregularity of the arterial pulse. Case VI. Jugulo-carotid and brachial tracings.

Lastly, it should be remembered that flutter is often the percursor of persistent fibrillation, and that many cases presenting the latter form of auricular action have probably passed through an unrecognised phase of flutter.

CHAPTER X

PROGNOSIS

As only a limited number of cases have hitherto been recorded, and as most of them were under observation for only a short period of time, the statistical evidence on which to base a prognosis is necessarily scanty. Certain general conclusions, however, may be drawn from a survey of the recorded cases.

In one group of cases the normal cardiac rhythm is eventually restored, and either the patient makes an apparently complete recovery and remains well for several years, or, failing that, his general condition is greatly improved. The normal rhythm was restored in twenty-two out of fifty-three cases. One patient (Case IV.) was perfectly well five months after a sharp attack of flutter. In Case XIX., the patient was seriously ill when the auricles were fluttering, yet the heart regained its physiological rhythm; three years later the patient was in good health, and had no trouble with his heart. In Case XXXII. auricular flutter passed into fibrillation, but four and a half years later the normal cardiac rhythm was found to be restored and the patient was earning his living. In Case VIII., the patient was in excellent health four years after a prolonged attack of tachycardia almost certainly due to flutter.

A second group, a large one, comprises the cases with paroxysmal attacks of flutter recurring from time to time over a period of several years. In Case XXXIX. attacks recurred occasionally during a period of two years. In Case XVIII. they recurred at intervals during at least eleven years. In Cases XXIII. and XXIV., in which auricular flutter was subsequently demonstrated, there had been a liability to paroxysmal attacks of tachycardia for thirty and thirty-eight years respectively.

A third group consists of cases in which auricular flutter persists more or less continuously for many weeks, months, or even years. Flutter persisted for four months in Case XIII., for six months in Case XXV., for nine months in Case XIV., for eighteen months in Case XVI., and for nearly five years in Case I. Even after the flutter has persisted

for years, however, the auricles may eventually regain their normal rhythm (see Case I.).

A fourth group, one of considerable magnitude, includes cases in which flutter is the precursor of a more or less persistent auricular fibrillation. This sequence was observed in Cases II., V., and XLVII.

A fifth group is constituted by cases in which a fatal issue ensued. In Cases III. and XXXVI. death occurred ten and nineteen days respectively after the first records of flutter had been obtained. In Case XII. seven weeks elapsed between the onset of flutter and the patient's death. In Case XXI. the patient died nineteen months after the onset of his illness. In Case XXIII. there had been paroxysmal attacks of tachycardia for thirty years before death. When the fatal issue supervenes it may be sudden, as in Cases VI., XII., XXI., and LIII., or gradual as in Cases XI., XXXV., and XXXVII.

The prognosis is more favourable when the heart and vessels are apparently healthy than when there is obvious disease of the myocardium or valves, or definite arterio-sclerosis.

Again, an initial attack of flutter will probably be milder and shorter than subsequent attacks. Indeed, the recovery from a first attack is probably invariably complete for a time at any rate, unless there be advanced disease of the myocardium, as in acute infective myocarditis or in the terminal phases of mitral disease.

During an attack of flutter the severity of the dyspnœa, the intensity of the cyanosis, and the urgency of other symptoms furnish the best guide to prognosis. Alternation of the pulse when the rate is only 120 per minute is usually an unfavourable sign ; but when the rate is 150 or more, alternation is not necessarily serious. Its absence when the pulse-rate exceeds 150, though probably indicating a comparatively healthy ventricle, does not necessarily imply a good prognosis (see Case III.).

When auricular flutter supervenes in patients with advanced chronic heart disease, the prognosis is particularly unfavourable, because the change almost certainly indicates the extension of inflammatory processes in the auricular walls, and also because the acceleration of the ventricles increases still further the insufficiency of these chambers. The flutter as a rule either persists for weeks or months, or passes into auricular fibrillation. In either case there is little probability of the normal rhythm being restored, and in certain instances the flutter and fibrillation are terminal events.

In a favourable case, once the attack has subsided and the normal

cardiac rhythm has been restored, the liability to recurrence of flutter is decidedly more pronounced if the patient has a grossly damaged heart than if his heart be apparently healthy, because in the former case there is the constant risk of fresh outbreaks of myocarditis. At this stage prognosis will depend in large measure upon the patient's habits and the nature of his occupation. The man or woman who can lead a quiet life, avoiding physical strain, and who is little subject to emotion and worry, has the better prospect of avoiding subsequent attacks.

CHAPTER XI

TREATMENT

Our first aim in the treatment of auricular flutter is the relief of the patient's urgent symptoms. This is usually effected when the flutter subsides and the physiological rhythm is restored, or when the ventricular rate falls to about the normal in spite of the auricles continuing in flutter. Further, we have to treat the causal condition whether it be cardio-vascular or nervous, infective or toxic, structural or functional; and lastly, we have to try to prevent the recurrence of the flutter.

1. *Treatment during an attack* should be directed to arresting the flutter, or, failing that, to lowering the excessive speed of the ventricles. Complete *rest* in bed is usually of great benefit and is always desirable. All unnecessary physical exertion or strain should be avoided. In some instances orthopnœa is so pronounced that the patient cannot lie down without aggravating the breathlessness; but as a rule, if there be cyanosis, dyspnœa, or other notable signs of heart-failure, the patient should be in the recumbent posture for the greater part of the day. It is often well, however, to permit him to sit up in bed while he is taking nourishment, and for about half an hour afterwards. Discomfort due to flatulence may thus be obviated. The patient should also be shielded in so far as is possible from all worry, excitement, and emotional disturbance which might stimulate the accelerator mechanism of his heart and thus tend to maintain the auricular flutter.

The *diet* should be light and digestible, but solids need not necessarily be withheld altogether. Nourishment should be given every two or three hours, so as to avoid overloading of the stomach at any one meal. Careful attention should be paid to the state of the bowels, any tendency to constipation, flatulence, or diarrhœa being corrected by appropriate dietetic and medicinal measures.

If the patient is restless or sleepless, *bromides* may with benefit be given in the evening. The calcium or ammonium salts are the most suitable. In one of Mackenzie's cases the prolonged administration of ammonium bromide in 20-grain doses thrice daily was helpful in pro-

moting sleep and in checking the more serious paroxysms of ventricular acceleration.

As the application of an *ice-bag* to the precordia is sometimes very soothing to patients with paroxysmal attacks of auricular fibrillation and other forms of "paroxysmal tachycardia," it might be well to give it a trial in cases of auricular flutter.

Digital compression of either the right or left vagus may be tried. In some cases of "paroxysmal tachycardia" this procedure has been effective in arresting an attack, and in auricular flutter we know that it often induces transient slowing of the ventricles. In well-authenticated cases of auricular flutter, however, the ventricular retardation resulting from vagus-compression is of very brief duration; the auricles meanwhile continue in flutter, and within a few seconds the high speed of the ventricles is regained. Consequently vagus-compression can hardly be regarded as having any real therapeutic value in auricular flutter.

The *drugs* that act most beneficially are digitalis and strophanthus. It is not necessary to administer these drugs in every case of auricular flutter. Case IV., for example, made a complete recovery without either drug being administered. But in most cases of auricular flutter the urgency of the patient's symptoms affords a definite indication for the use of one or other of these drugs. Their mode of action and their remarkable effects have been described in Chapter VIII. It will therefore suffice to state that the initial effect of these drugs is to lessen the rate of the ventricles. This often falls to one-half of its antecedent rate—for example, from 150 to 75 per minute—while the rhythm remains regular. Subsequently the auricular flutter may become replaced by fibrillation, and the rhythm of the ventricles then becomes wholly irregular, but their rate is usually not excessive. In some instances the normal rhythm is restored after the digitalis has been withdrawn; but this successful result is by no means constantly attained, and we must admit that the drug often fails to "make hearts beat our time that flutter false."

Although in some instances the heart with fluttering auricles is highly responsive to digitalis, it is often necessary to give this drug in considerable doses for several days before the ventricular rate falls and the patient obtains relief. Small doses, such as five minims thrice daily, may be of no avail. Twenty minims of digitalis tincture should be given thrice daily, and if the ventricular rate is not retarded in the course of three or four days the daily dose should be increased to one and a half drachms. The tincture is usually effective, but other preparations may be tried. Nativelle's granules ($\frac{1}{240}$ gr. of digitaline, thrice

daily) were given with benefit in Cases V. and XIX., digalen was used in Cases XV. and XVI., and digipuratum in Case XLII. If digitalis is to be administered by the mouth, it probably matters little which preparation be employed.

The drug should be given steadily until, the ventricular rate having fallen to about normal, the patient's urgent symptoms are relieved. At this stage the auricles, as a rule, are still in flutter. One of two courses may then be followed. The daily amount of the drug may be reduced until the minimal dose that suffices to restrain the ventricles is being given, and this dose is continued for days, weeks, or months until the flutter ceases. The alternative procedure is to maintain a large dose of digitalis until the auricles pass into fibrillation, and when this event supervenes to withdraw the drug altogether for some days in the hope of the normal cardiac rhythm being restored. In most cases it is neither advisable nor desirable to withhold the drug completely for many days, because the ventricular rate is apt to become too frequent, and consequently distressing symptoms are liable to recur. Yet a dose greater than is sufficient to hold the ventricles in check is equally undesirable, for the larger the dose of digitalis the less likelihood is there of the auricular fibrillation becoming replaced by a normal rhythm.

Should we deliberately push the dose of digitalis until the auricles are in fibrillation, or should we be content with retarding the ventricles while the auricles continue in flutter? Two further problems arise, namely, what prospect is there of the auricular fibrillation that succeeds flutter being replaced by a normal rhythm, and how long may the latter be maintained? The normal rhythm was restored in only fifteen out of thirty cases treated with digitalis, and in one instance the normal rhythm was apparently maintained for three years. Such a satisfactory result, however, is exceptional, and even after the normal rhythm has been restored it is liable to be interrupted from time to time by paroxysmal attacks of flutter or fibrillation. Again, we know that once auricular fibrillation is induced there is a distinct tendency for it to persist. This is a most undesirable event. Further, we know that even although the auricles continue in flutter the patient may remain wonderfully free of all distressing symptoms provided his ventricular rate be not unduly high. In Case II., for example, the patient felt perfectly well when the rate of his ventricles was one-fourth of the auricular rate. Under such circumstances the dose of digitalis should not be increased with the object of inducing auricular fibrillation. On the contrary, the dose should not be larger than is sufficient to restrain the ventricles. In

each case the dosage must be determined by the patient's general condition and symptoms, and in some cases it may be advisable to continue the administration in small doses for a long period, possibly for years (see Case XVIII.).

In spite of the ventricular rate having fallen, the patient's general condition may not be satisfactory. He may still be cyanosed, breathless, and dropsical. Under these circumstances the digitalis should be continued in considerable doses, and if there be no contraindication it may with advantage be combined with caffeine, diuretin, theocine, potassium acetate, or other diuretics. In my experience the combination of digitalis with potassium iodide often acts remarkably well in cases of chronic interstitial myocarditis. In one case of auricular flutter this combination was certainly helpful. Mackenzie[1] is undoubtedly correct in maintaining that when digitalis is administered in therapeutic doses it does not raise the arterial pressure. Some cases of auricular flutter, nevertheless, show a decided tendency to arterial spasm, the hypertonus described by Russell.[2] This tendency should be corrected by attention to the patient's diet, by the administration of an occasional mercurial purge, and, if necessary, by the use of nitrites.

The action of strophanthus (Cases III., XXIV., and XXV.) and of squill (Cases I. and XXXII.) has been investigated in only a few cases of auricular flutter, but it appears to be similar to that of digitalis.

When auricular flutter supervenes late in the course of mitral disease, or when flutter is associated with grave and urgent signs of heart failure, either strophanthin or some preparation of digitalis should be given intravenously. In Case III., with auricular and ventricular rates of 320 and 160 per minute respectively, 0·001 gramme of strophanthin (Boehringer) was given intravenously with great benefit. Twelve hours later the auricles were in fibrillation, while the ventricular rate had fallen to about 96-104 per minute, and the patient's general condition had improved. On the following day the normal rhythm of the heart was restored, and the rate was only 90 per minute. In another case the intravenous administration of strophanthin arrested a paroxysmal attack which was probably auricular flutter. When the patient, an old lady of seventy-four, was first seen, she was apparently dying of heart failure. The extremities were cold, the pulse-rate was 150 per minute, and the beats were very feeble, but they were rhythmic except for an

[1] Mackenzie, J., "Digitalis," *Heart*, Lond., 1910-11, ii., 273.

[2] Russell, W., *Arterial Hypertonus, Sclerosis, and Blood-Pressure*, Edin. and Lond., 1907.

occasional "intermission." A few hours after 0·0005 gramme of strophanthin had been given intravenously the patient began to improve, and a fortnight later her pulse was rhythmic, at a rate of 80 per minute. So satisfactory was the recovery that the patient was able to travel to London about two months later. Subsequently the attacks of heart failure recurred and became more frequent, and the patient died seventeen weeks after the initial attack.

2. *Treatment of the Causal Condition.* — In the majority of cases, auricular flutter is a result of sub-acute or chronic inflammation of the auricular musculature. This is often dependent upon disease of the valves or of the pericardium, or still more frequently upon arteriosclerosis. All these causal factors should be borne in mind in treating a case of auricular flutter. A history of rheumatism or of syphilis may yield a clue to appropriate and successful treatment.

3. *The Prevention of Recurrent Attacks.* — Success or failure in the treatment of each case, during an attack or after it has subsided, is largely dependent upon our ability to treat the underlying cause. Although a pre-existing mitral stenosis cannot be abolished, or thickened coronary arteries be replaced by healthy vessels, much may be done to check the further progress of the disease. The patient should be advised regarding his general mode of life. He should be directed to relinquish his arduous occupation for one that entails little physical or mental strain. The amount of physical exercise or intellectual work that he may undertake daily without risk to his health should be indicated, and any tendency to indiscretion in food, drink, or tobacco should be corrected. By attention to any obvious or latent sources of infection, whether in the alimentary tract or elsewhere, the risk of the myocardium becoming re-infected may be lessened. By these measures we strive to prevent the paroxysmal attacks from recurring, and even although in some cases we realise that our efforts are foredoomed to failure, in others we may hope for no small measure of success.

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